Vomiting in Infants

A 2 months old male, was delivered at term with birth weight 3,100 g presented with recurrent vomiting that started at age of 2 weeks. He received IV fluid therapy several times.

O/E, he looks lethargic, weight 3,250 g., dry tongue

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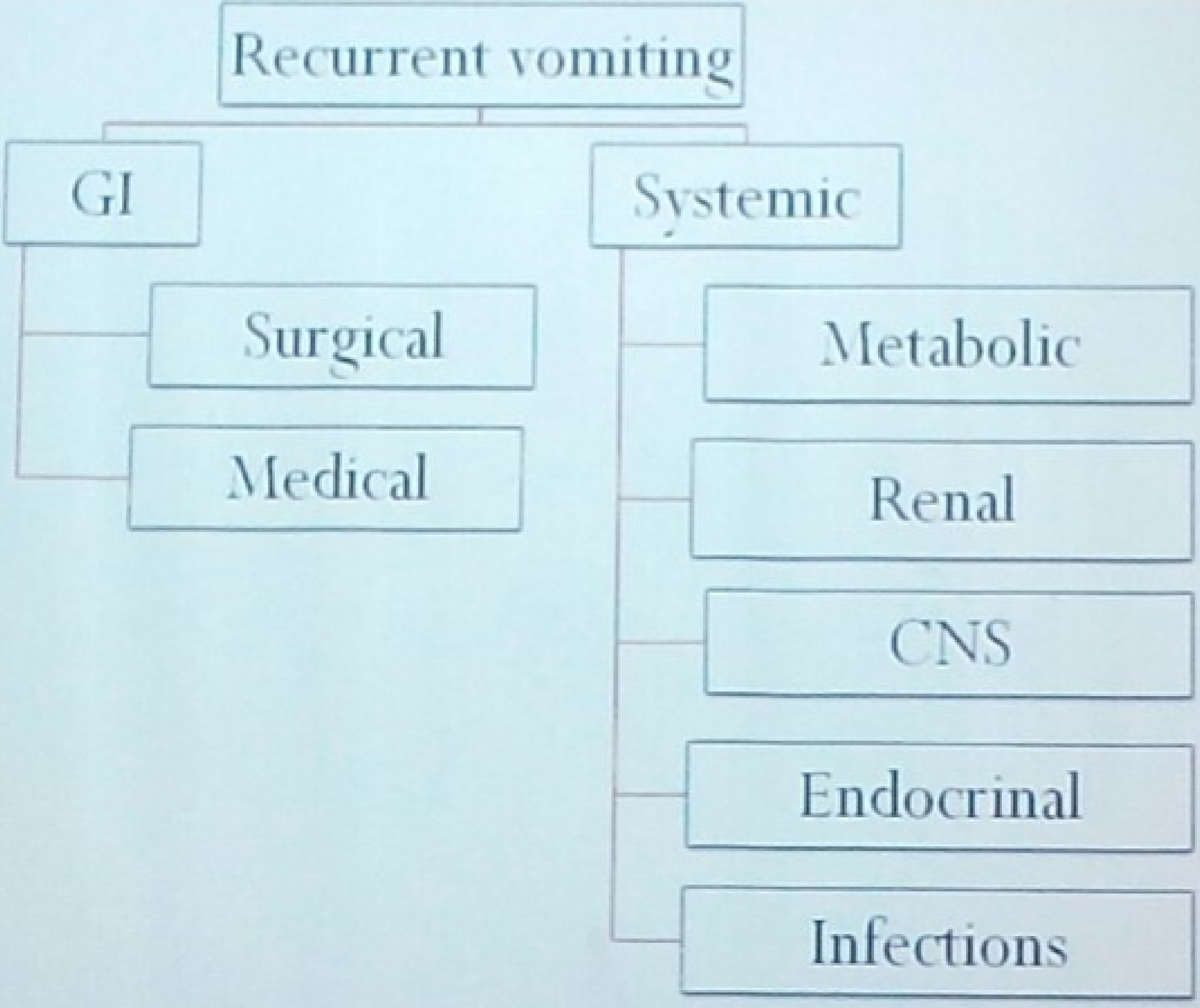
O/E, he looks lethargic, weight 3,250 g., dry tongue

A) What are the possible causes ??

B) What are investigations (urgent & routine)??

C) What are important points in history & examination

Vomiting is a non-specific symptom with a wide spectrum of causes



- Recurrent vomiting
  - GI
    - Surgical
    - Medical
  - Systemic
    - Metabolic
    - Renal
    - CNS
    - Endocrinal
    - Infections

Systemic (Extra GI)

1. IEM: organic acidemias, urea cycle defects
2. Infections ?? Immune def (UTI, OM, Pneumonia, sepsis)
3. Endocrinal: CAH, DKA
4. Renal: tubular defects (Barter, RTA), renal failure, obstructive uropathy
5. CNS causes: Increased ICT (hydrocephalus), Subdural hematoma, CNS anomalies

I. GI

A) Surgical

1. CHPS
2. **Other rare causes of gastric outlet obstruction:**

   prepyloric membrane.
3. **Mal-rotation with intermittent volvulus**
4. **Cardiac achalasia**

GI causes of vomiting

1. **GERD (with or without HH)**
2. **Food allergy (CMPA, Multiple)**
3. Eosinophilic GIDs
4. Inflammatory bowel disease
5. Peptic ulcer disease
6. Pancreatic disorders

A 2 months old male, was delivered at term with birth weight 3,100 g presented with recurrent vomiting that started at age of 2 weeks. He received IV fluid therapy several times.

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A) What are investigations??

Emergency investigations

Laboratory

1. Electrolytes (Dehydration, CAH, CHPS, RF)
2. Blood glucose (DKA, CAH)
3. ABGs
4. Kidney function tests
5. Liver function tests
6. Serum ammonia

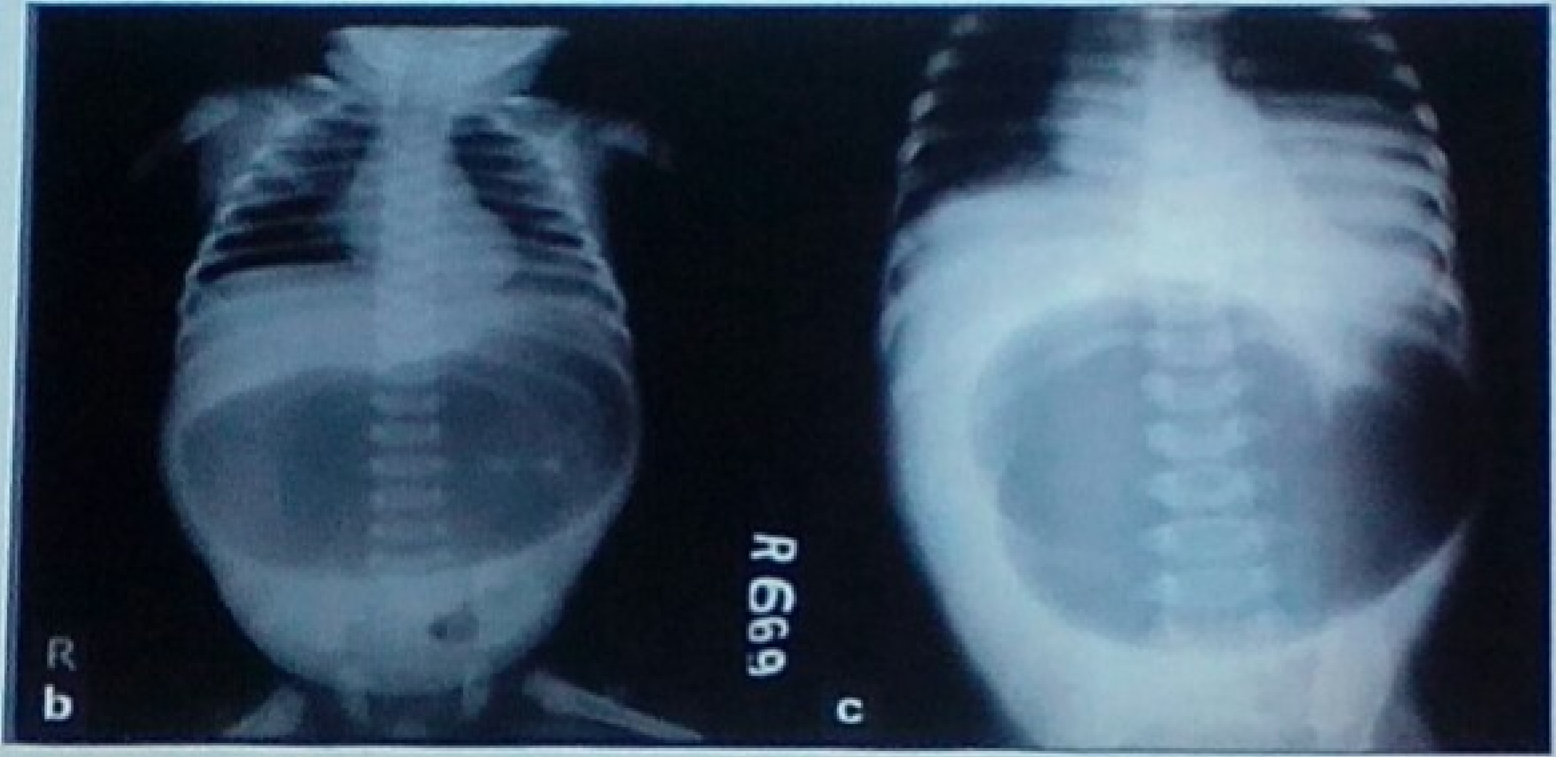
Value of electrolytes

	Na	K	Cl
CAH	Decreased	Increased	Decreased
CHPS	varaible	Decreased	Decreased
Renal failure	Variable	Increased	variable
RTA	Decreased	Decreased	Increased
Bartter $	Decreased	Decreased	Decreased

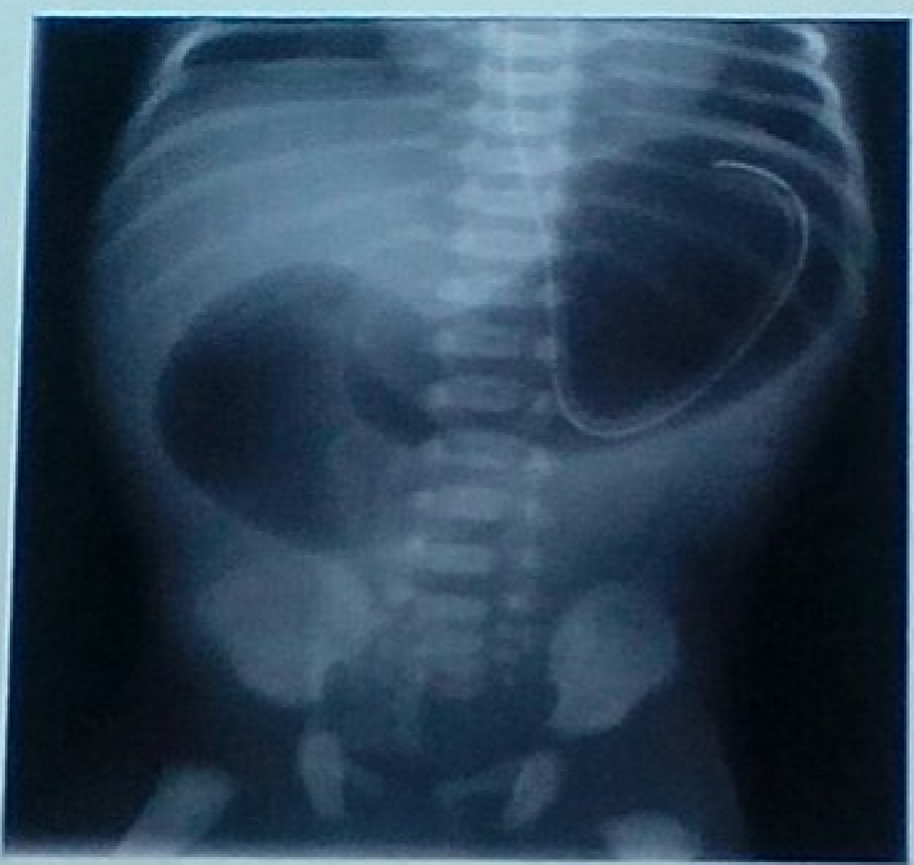
Value of ABGs

Metabolic Alkalosis	Metabolic Acidosis
CHPS	IEM: organic acidemias
Urea cycle defects (respiratory alkalosis)	RTA
Bartter disease	RF
	DKA
	GE

Radiological; Plain X ray abdomen

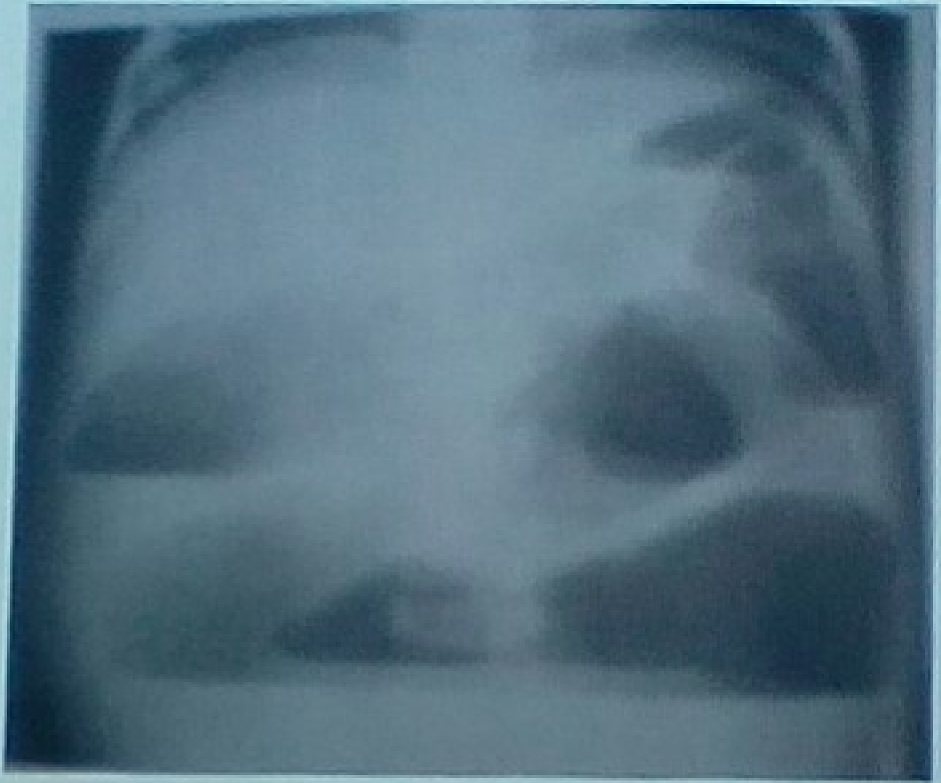


Radiological; Plain X ray abdomen



Plain X ray: Duodenal obstruction

Radiological; Plain X ray abdomen



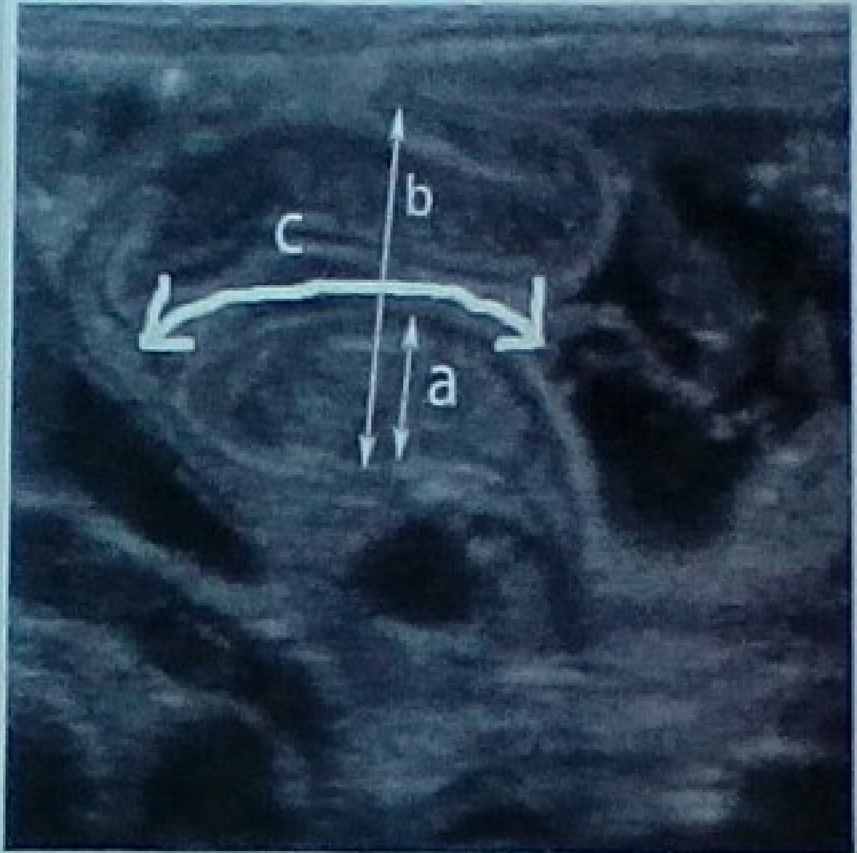
Abdominal US

CHPS:

Thickness greater than 4 mm

Length greater than 14 mm.

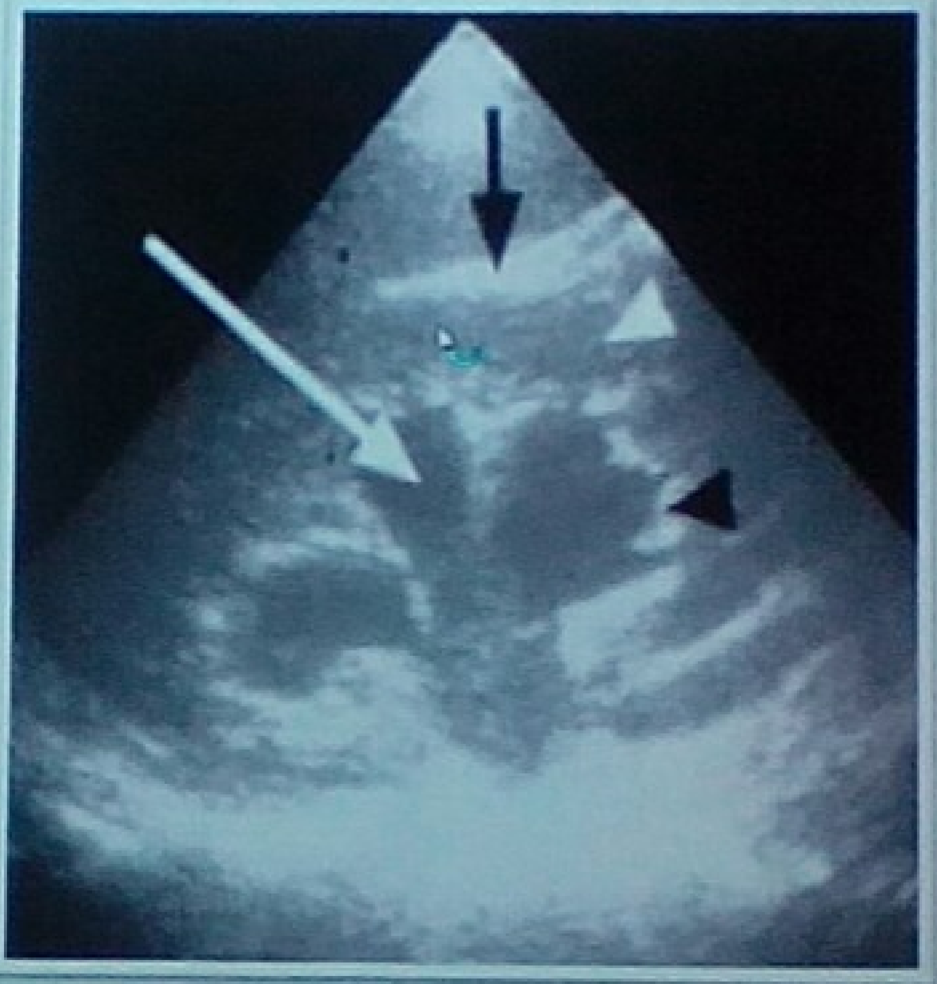
sensitivity of approximately 95%.



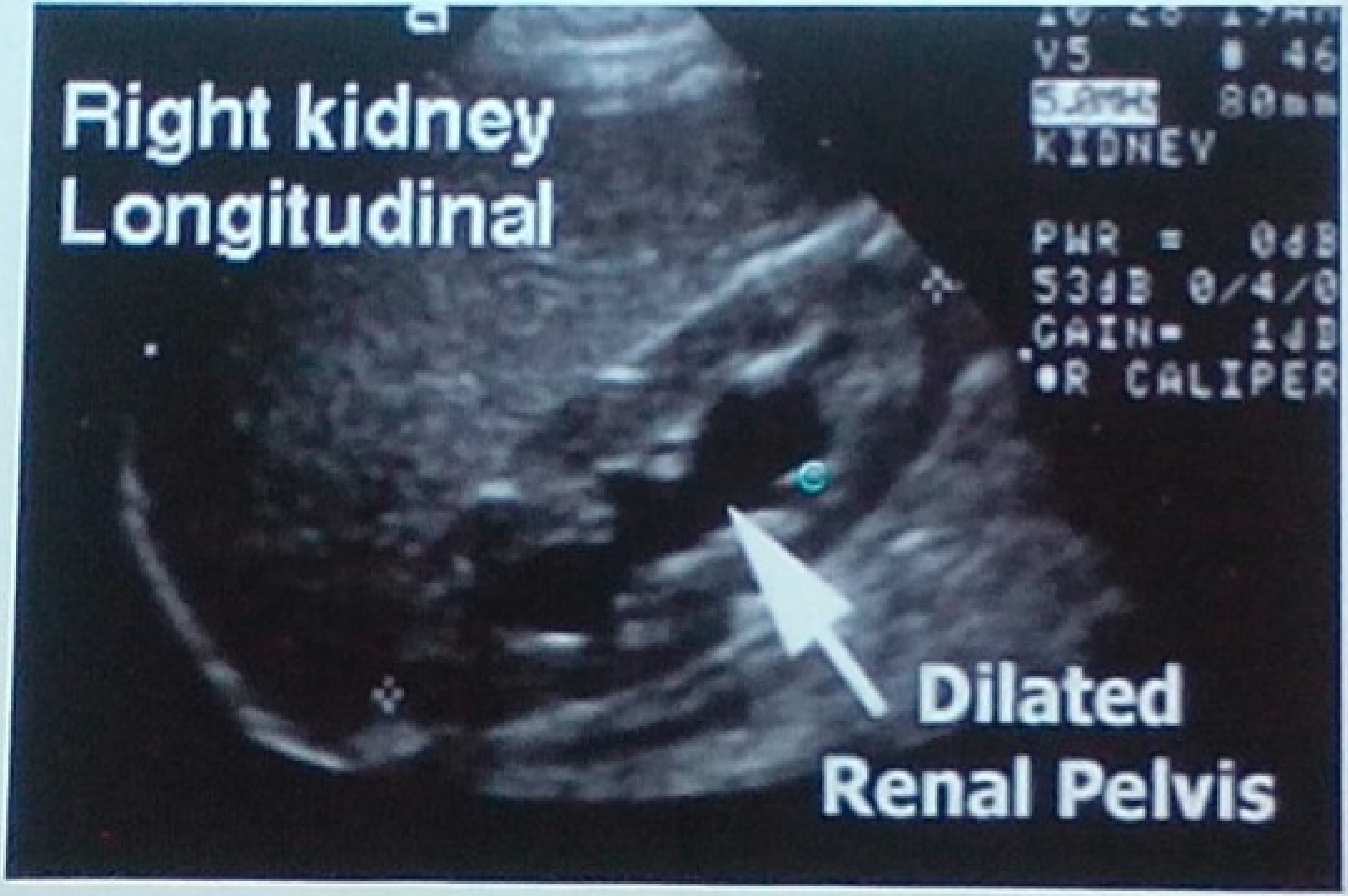
Abdominal US

Hydronephrosis

Dilated Calyces



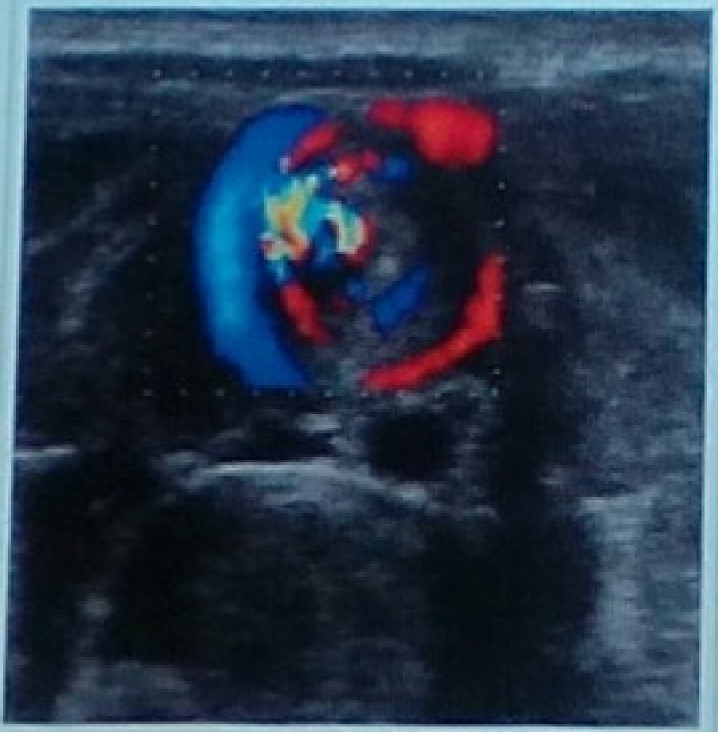
Abdominal US



Right kidney Longitudinal

Dilated Renal Pelvis

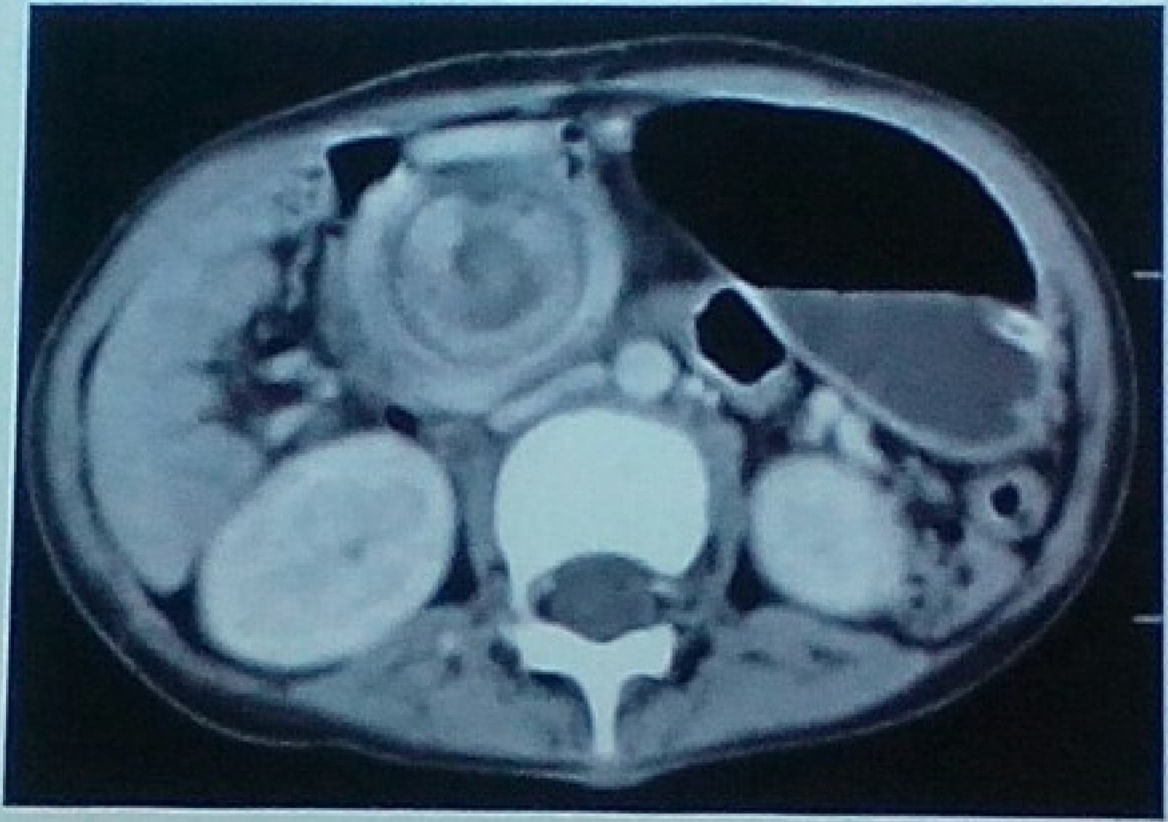
Abdominal US

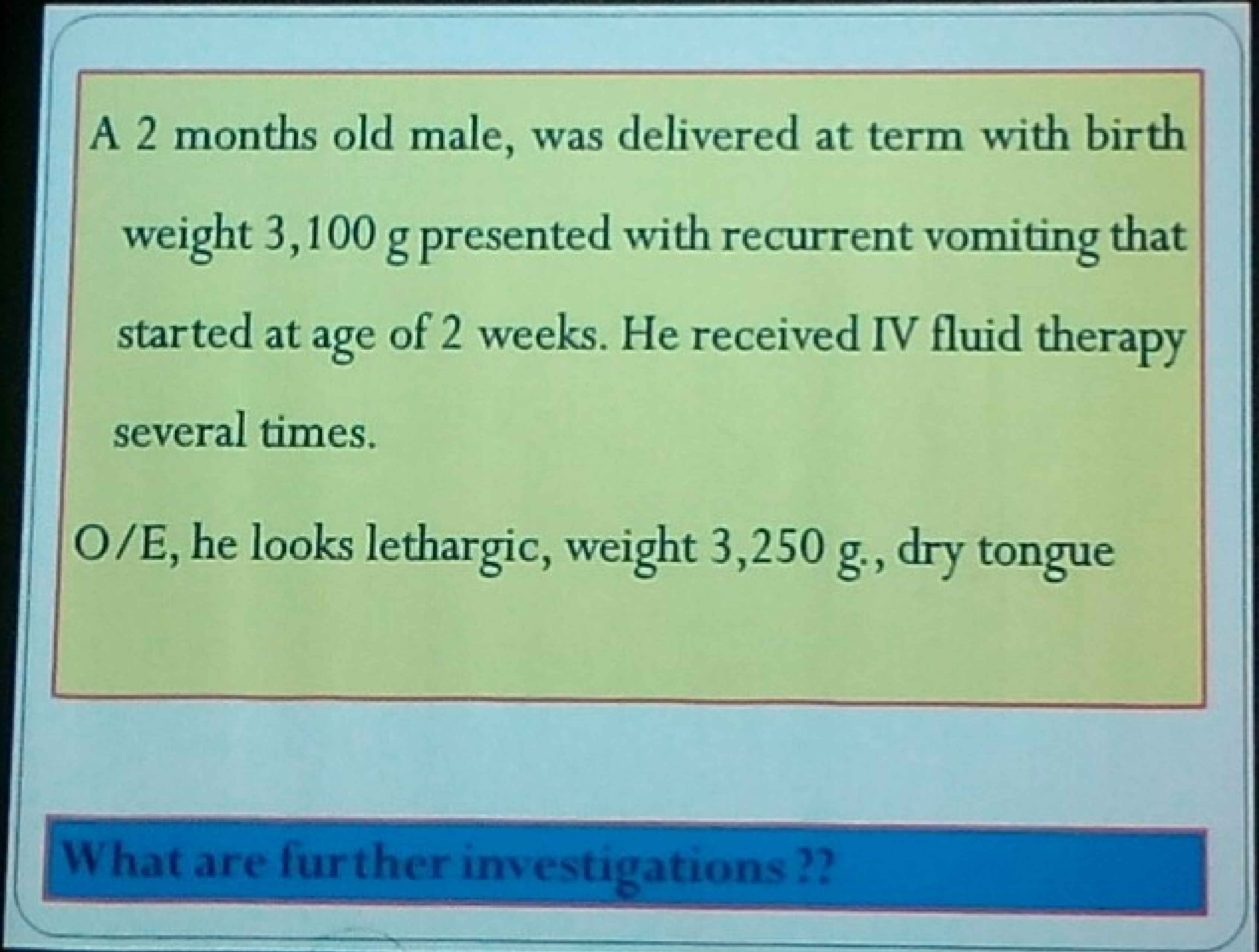


Whirpool sign

Whirpool

Midgut volvulus





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O/E, he looks lethargic, weight 3,250 g., dry tongue

What are further investigations ??

Further Investigations

Sepsis screen: CBC, CRP

Radiologic:

- Barium meal & follow through
- CT Abdomen
- CT brain

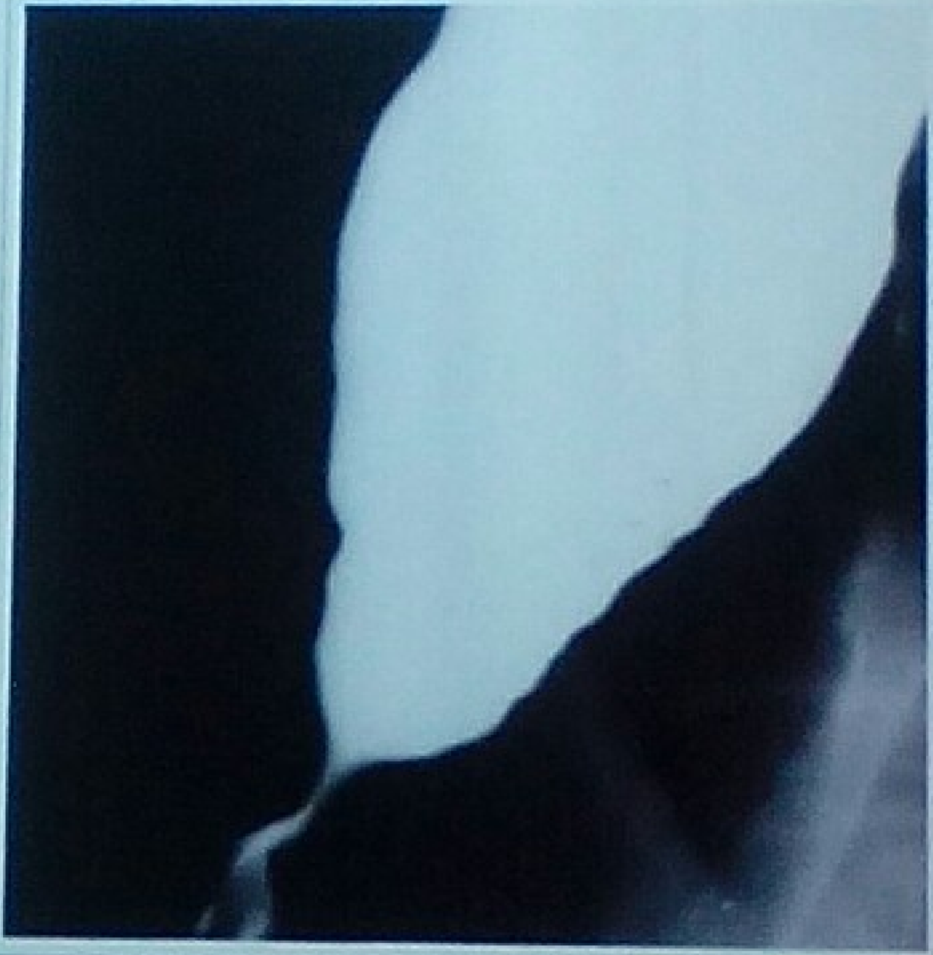
Metabolic screening:

Urine analysis & fundus examination

Upper GI series

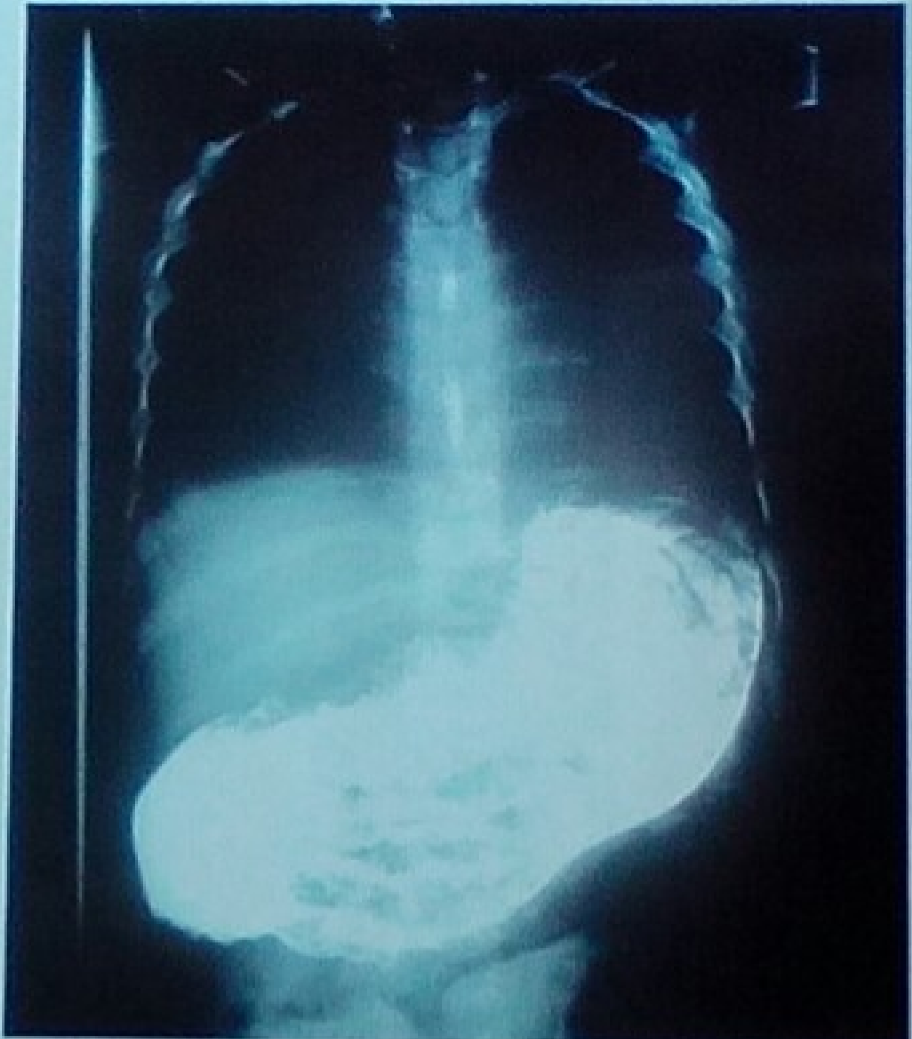
Cardiac

Achalasia

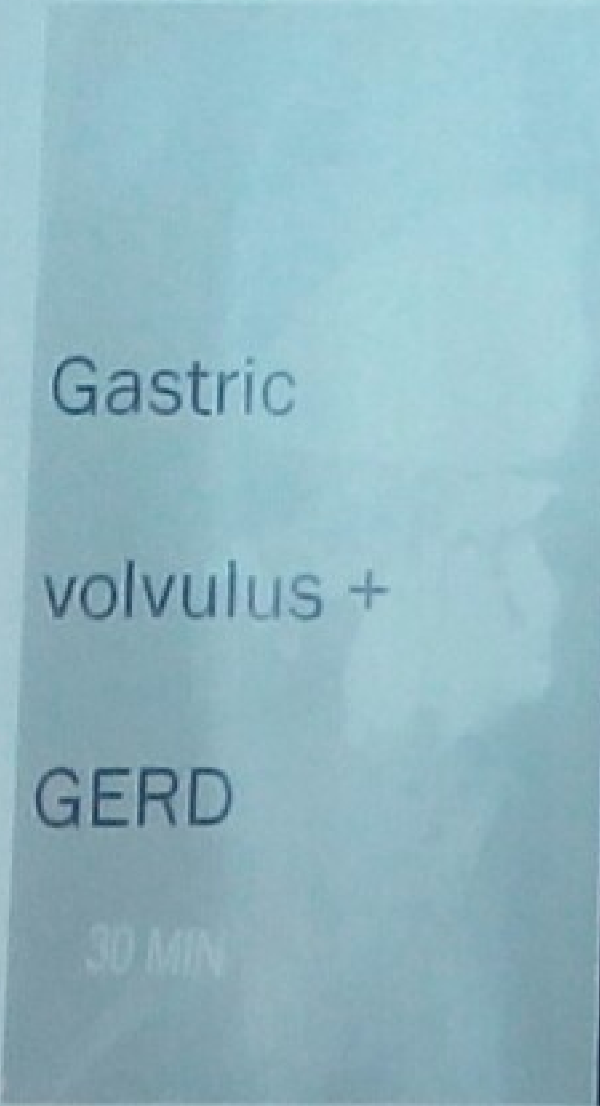


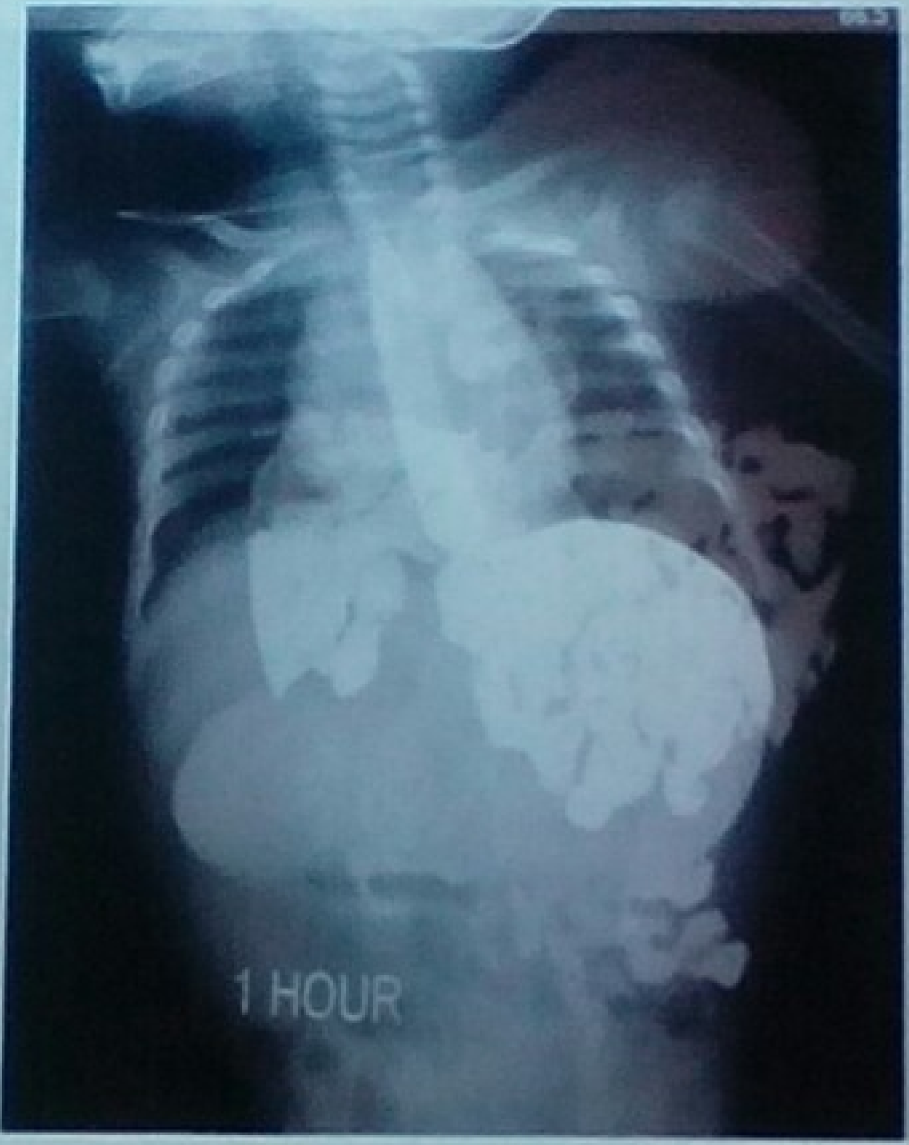
Upper GI Series

Pyloric obstruction+
dilated stomach

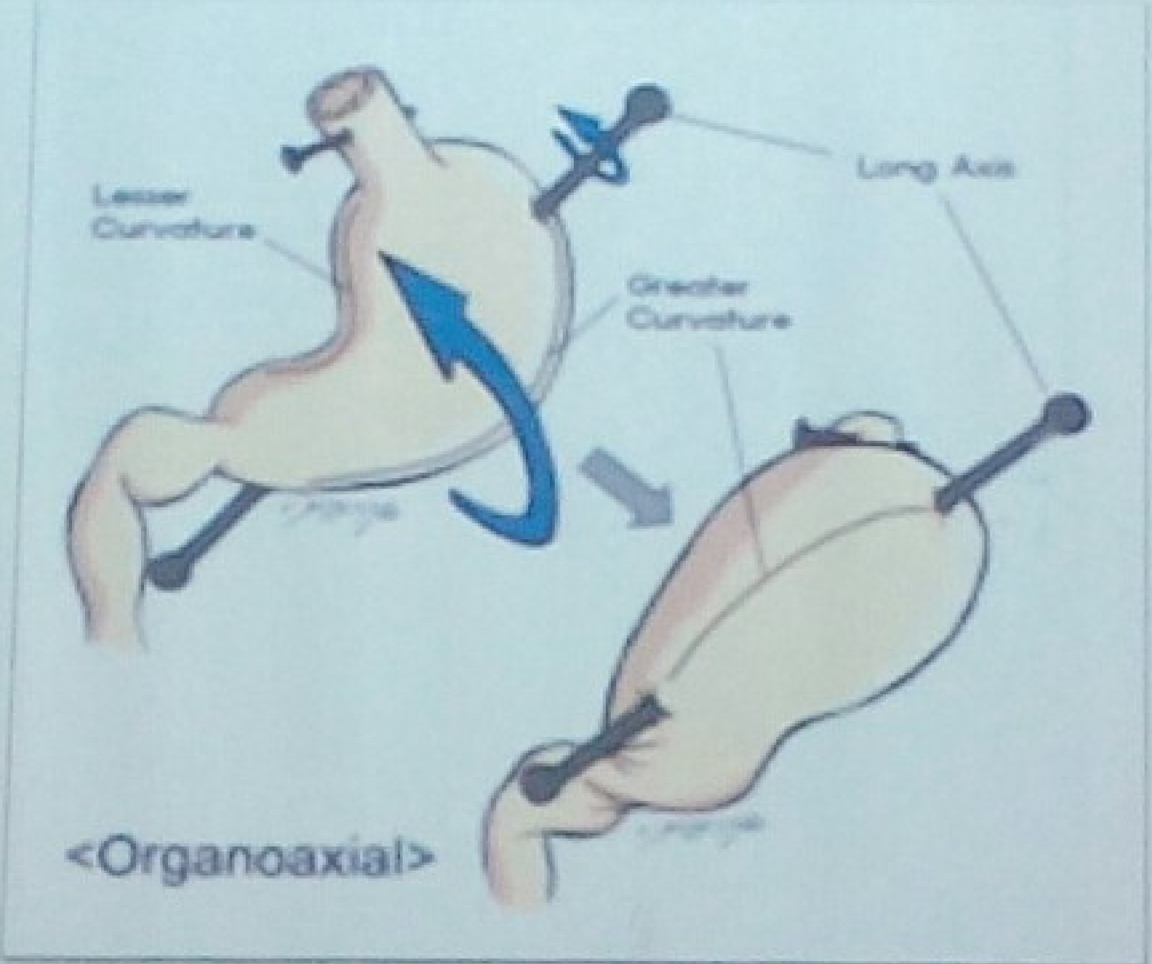


Gastric volvulus + GERD

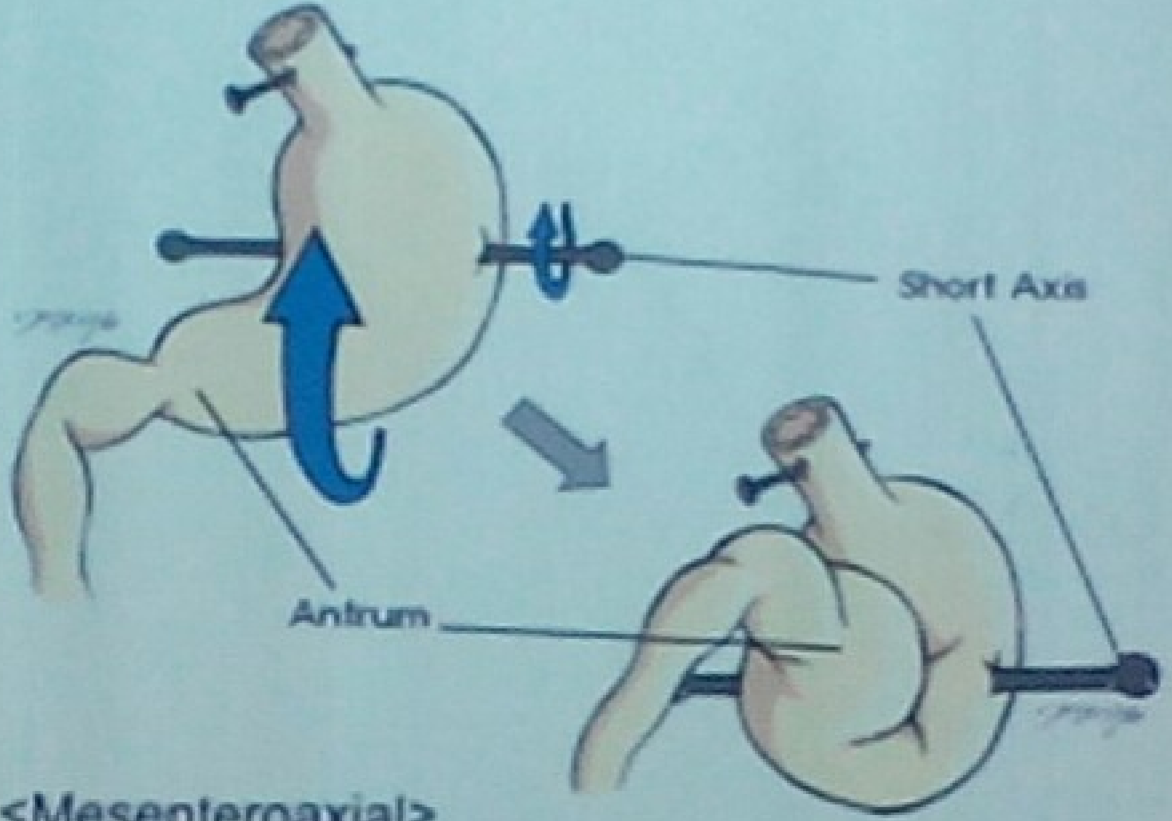




Gastric volvulus



Gastric volvulus



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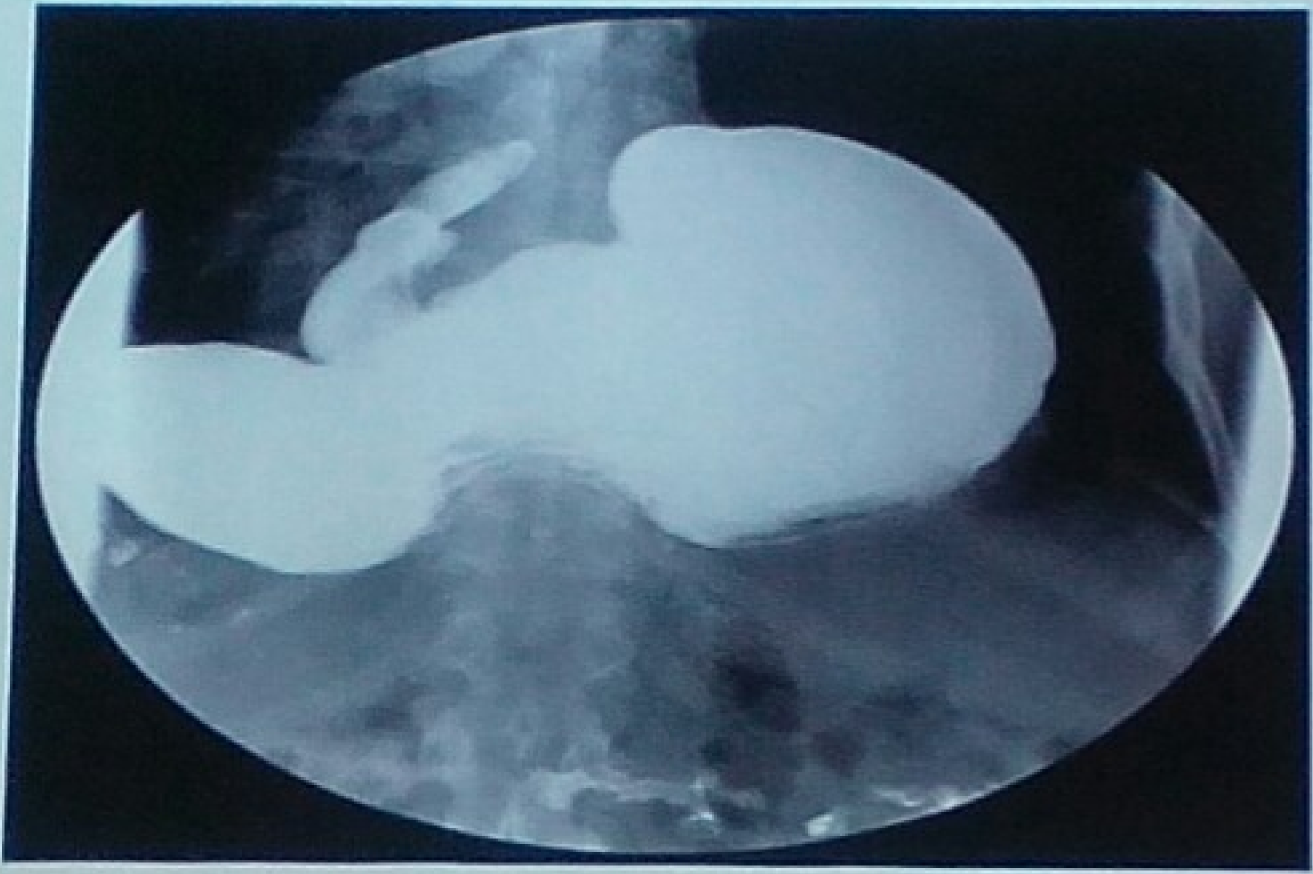
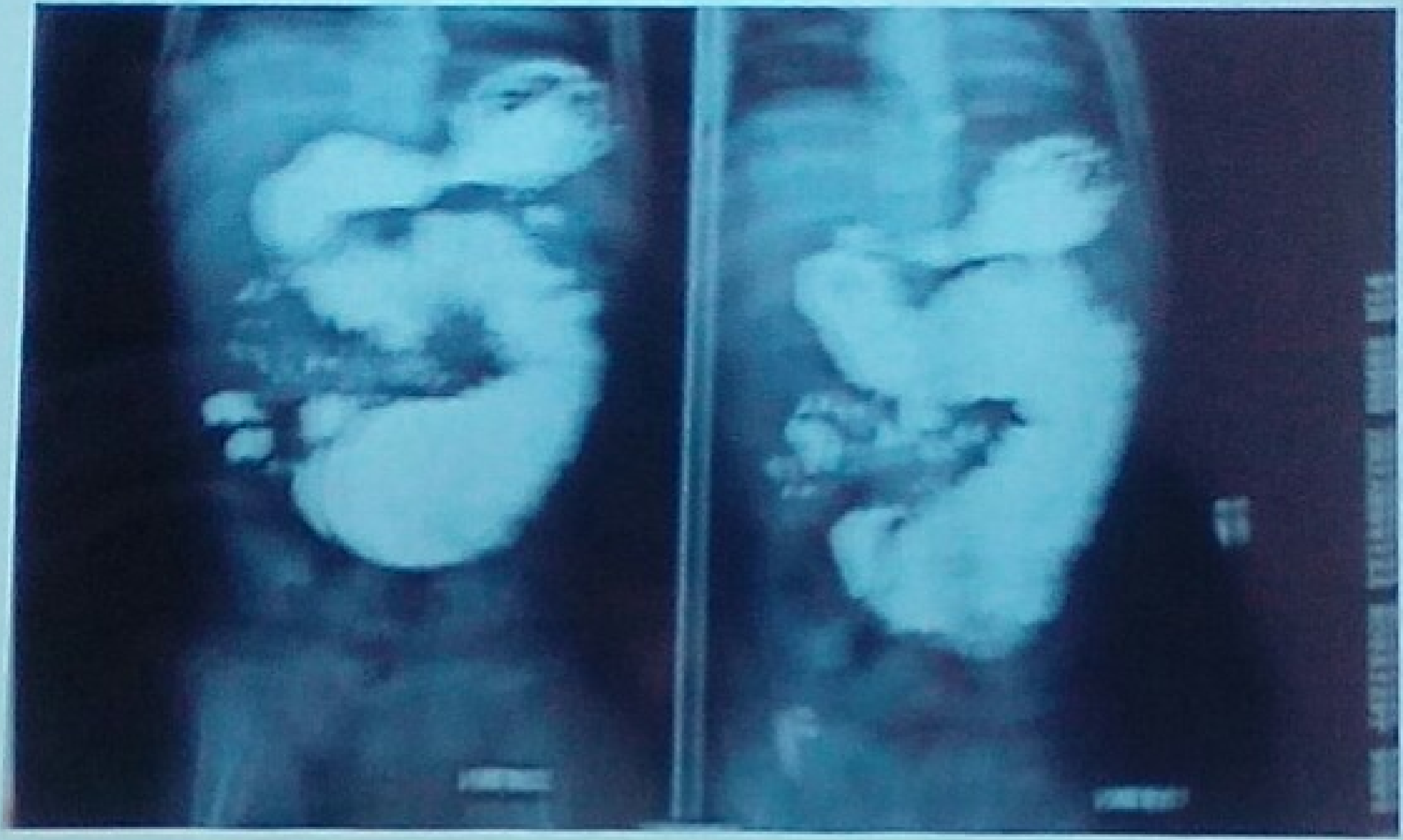
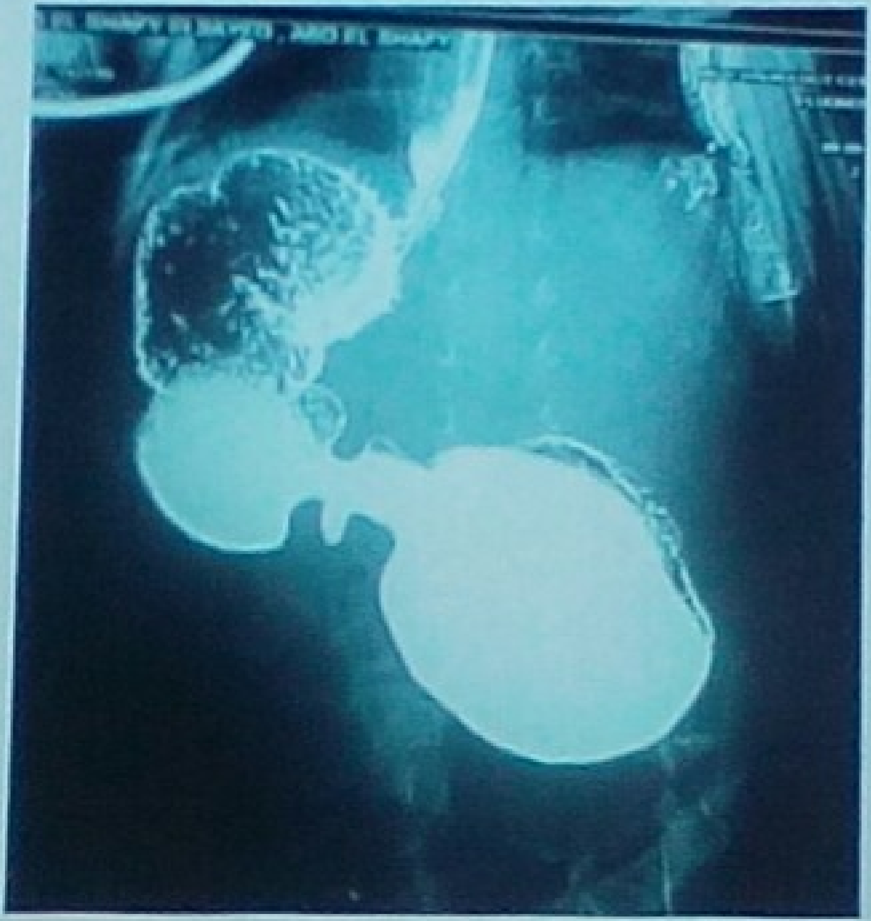
Gastric volvulus



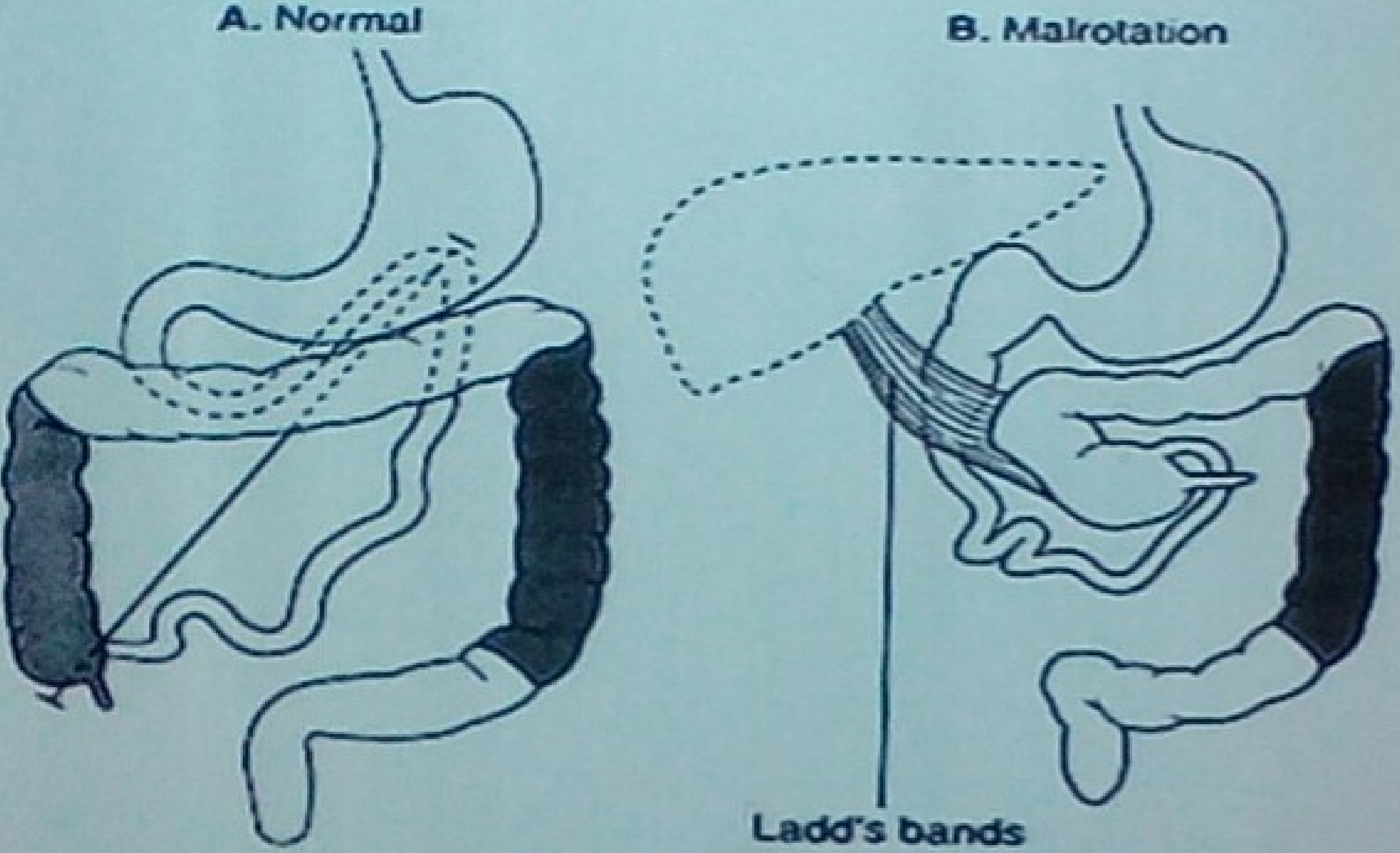


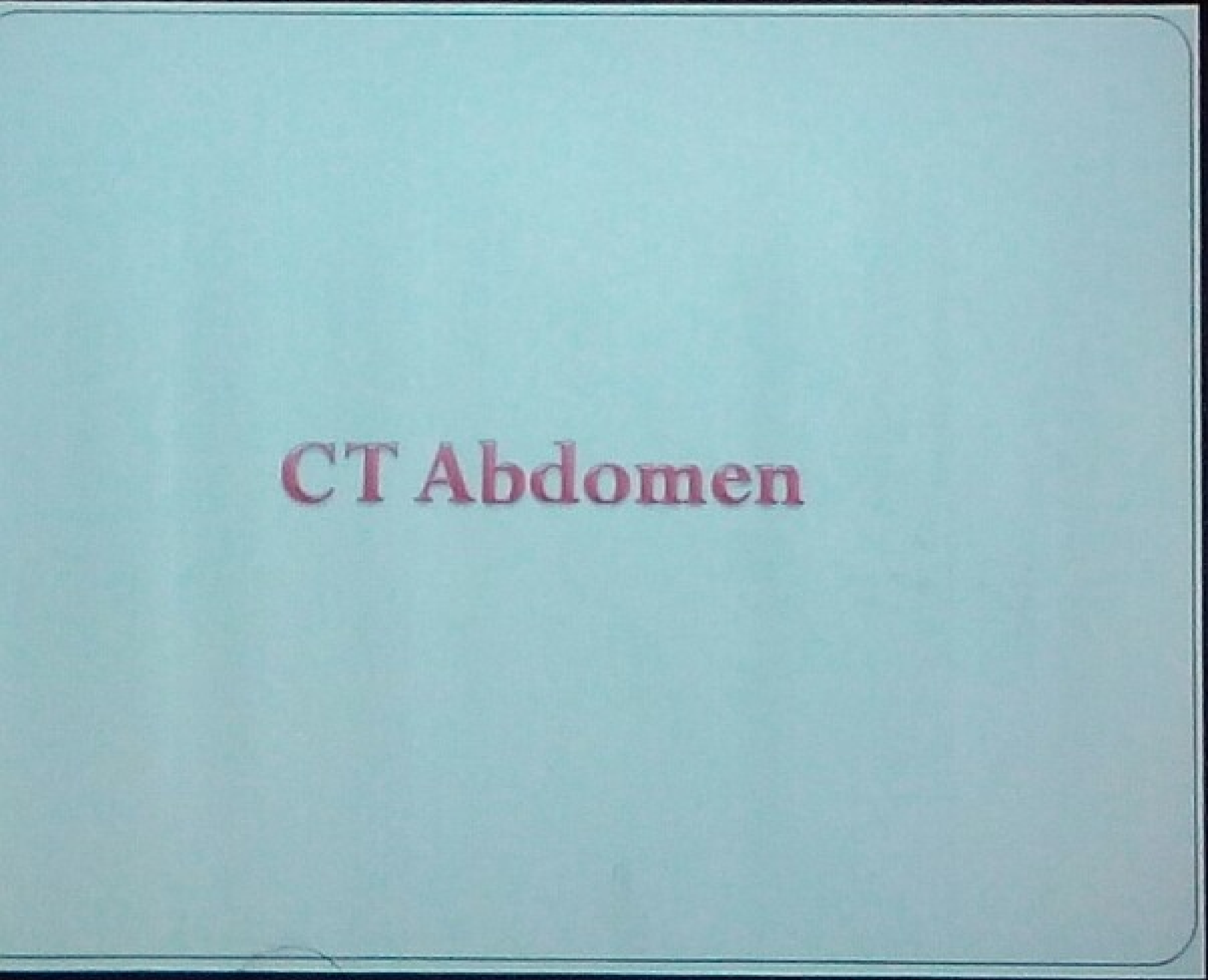
Wind Sack anomaly



Duodenal obstruction

Midgut malrotation





CT Abdomen

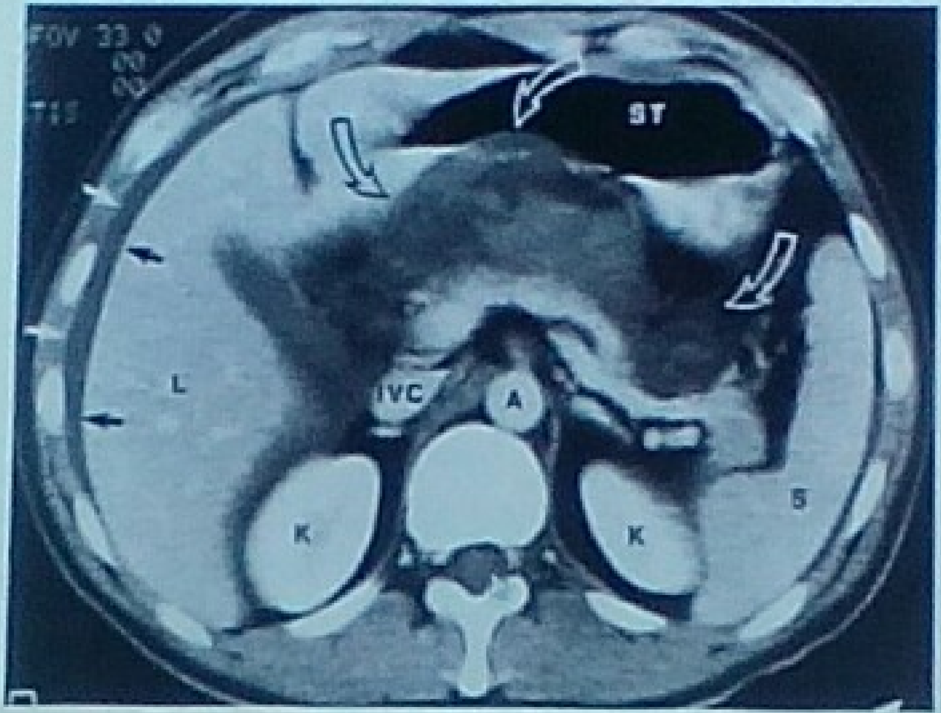


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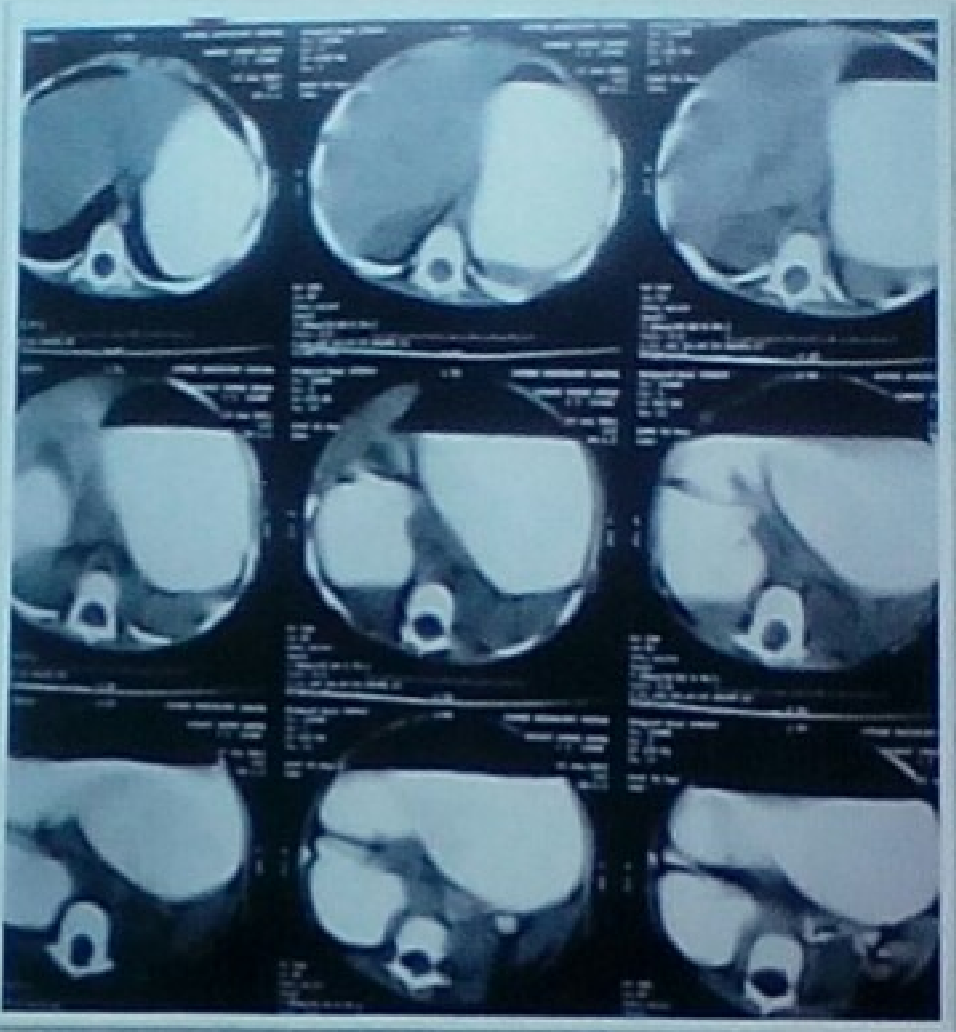
Figure 332-1 Acute pancreatitis. Computed tomography (CT) through the body of the pancreas demonstrates a halo of decreased attenuation around the pancreas that represents a peripancreatic zone of edema and fluid *(curved arrows)*. Note the pancreatic ascites most obvious lateral to the liver *(small arrows)*. If intravenous contrast was administered before the CT scan, the inflamed

CT Abdomen

Dilated stomach &

Duodeneum.

Dudenal diaphragm



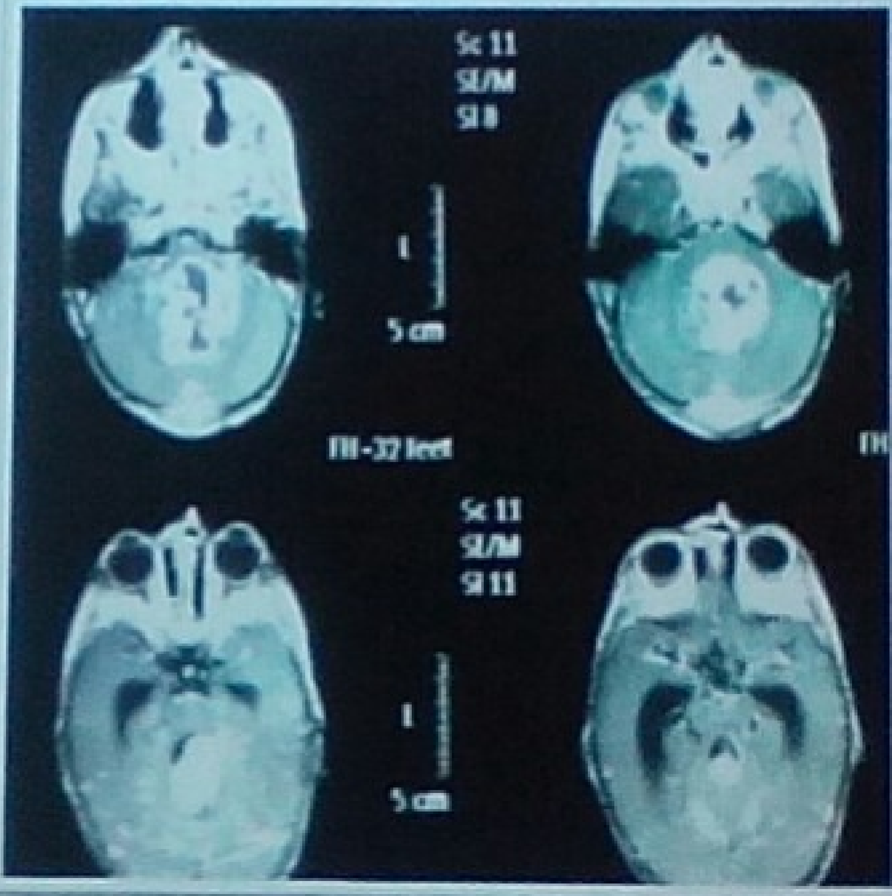
CT brian:

A 4 year old boy, with history of vomiting for one months that did not respond to PPI therpay and was refered for endoscopy

Detailed history revealed mild headache

O/E: He was lethargic hypotonic.

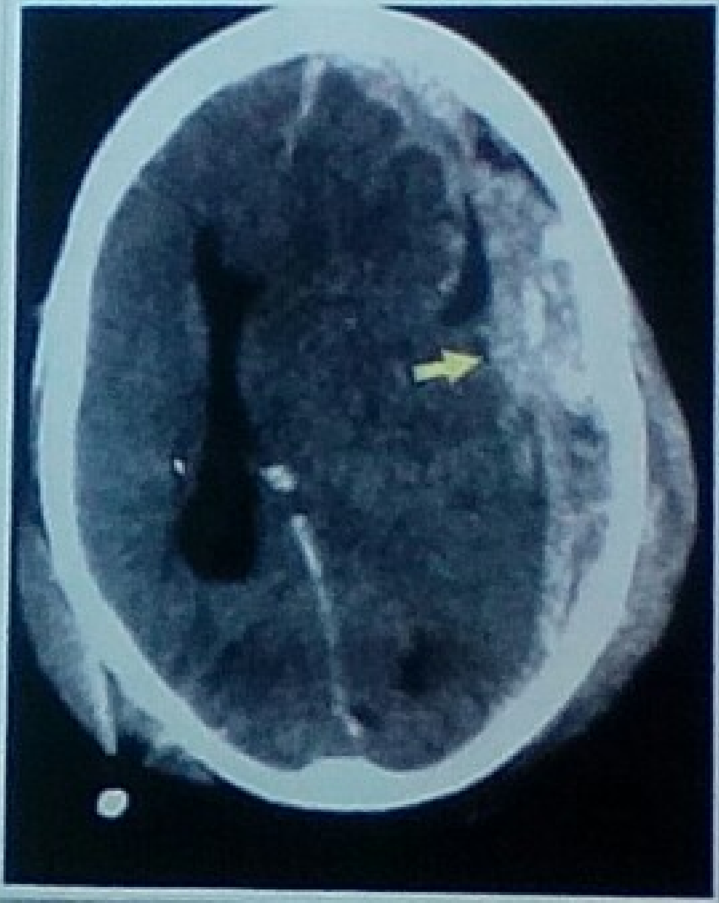
No epigastric tenderness.



CT Brain

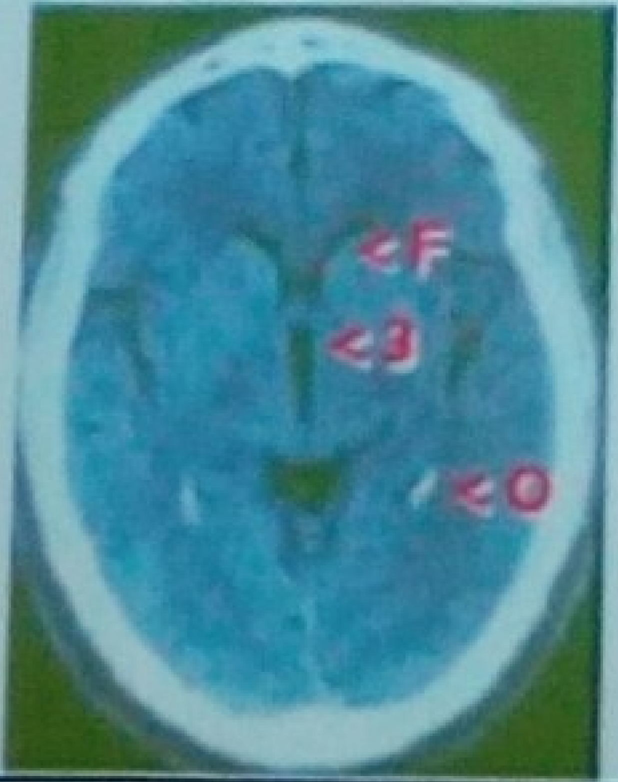
Subdural

hematoma

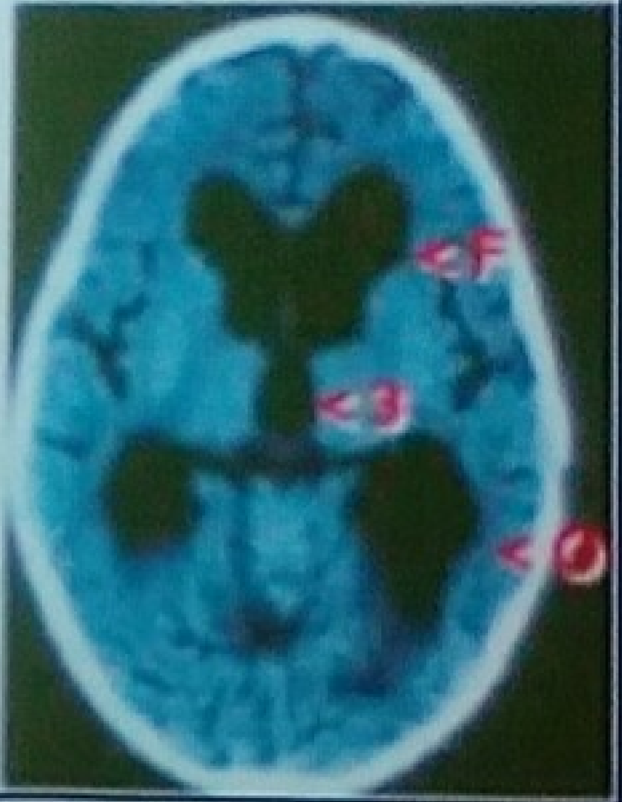


CT Brain

normal CT

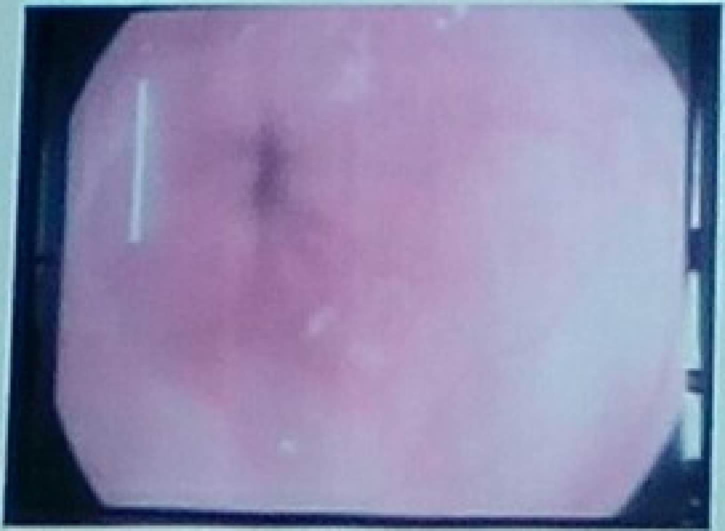


hydrocephalus



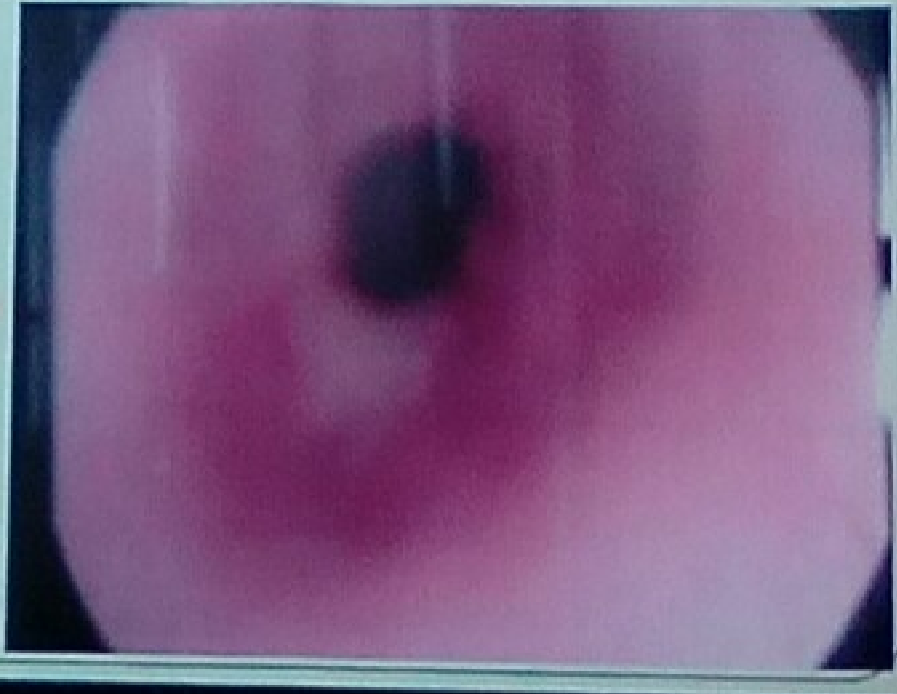
?

Endoscopy

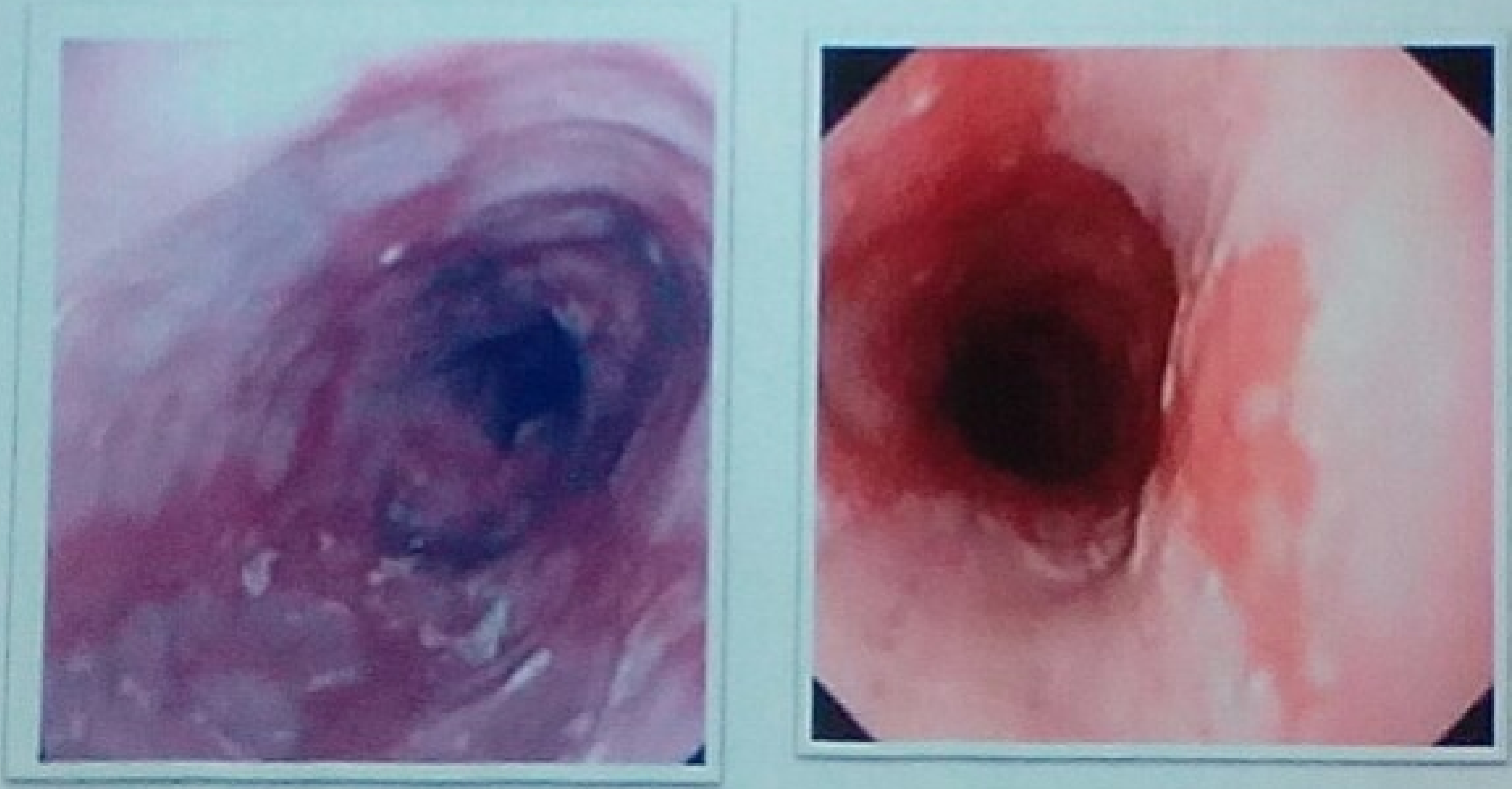


Competent
Cardia

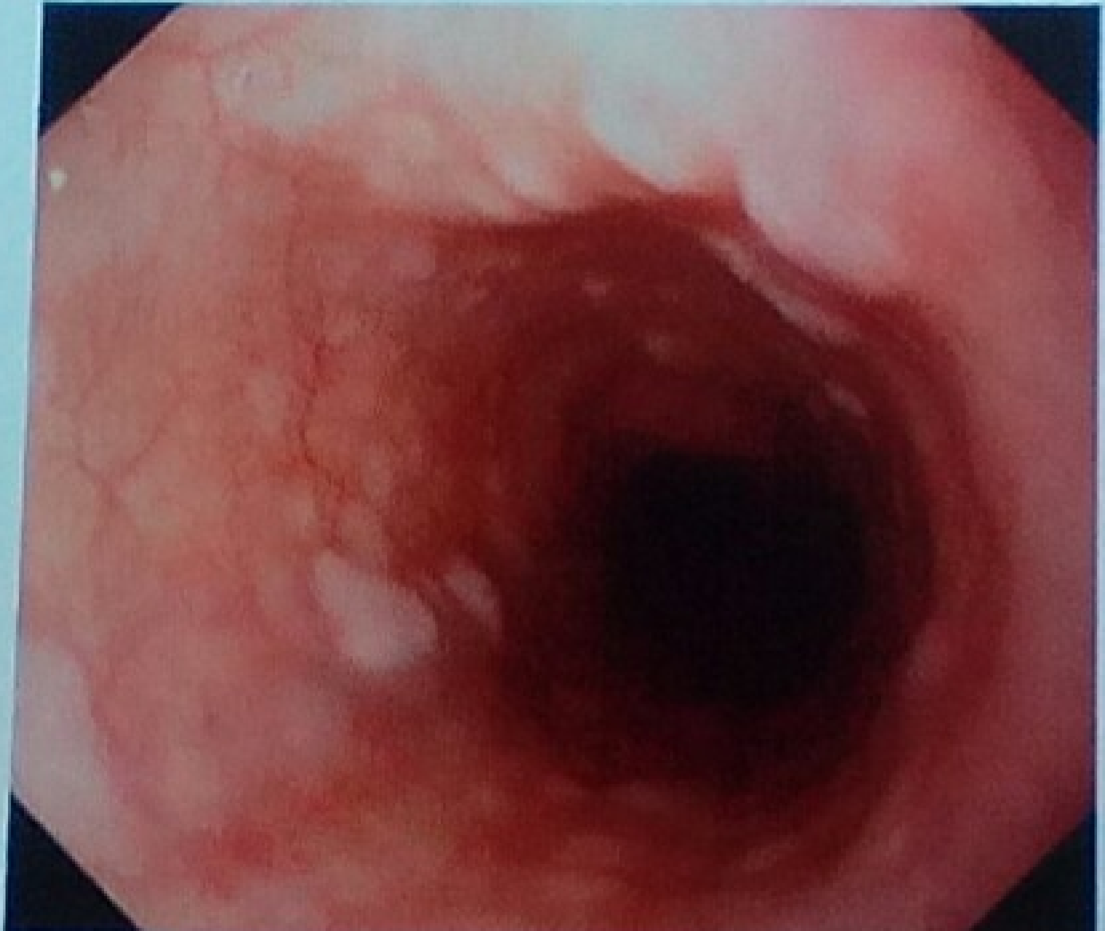
Incompetent
Cardia



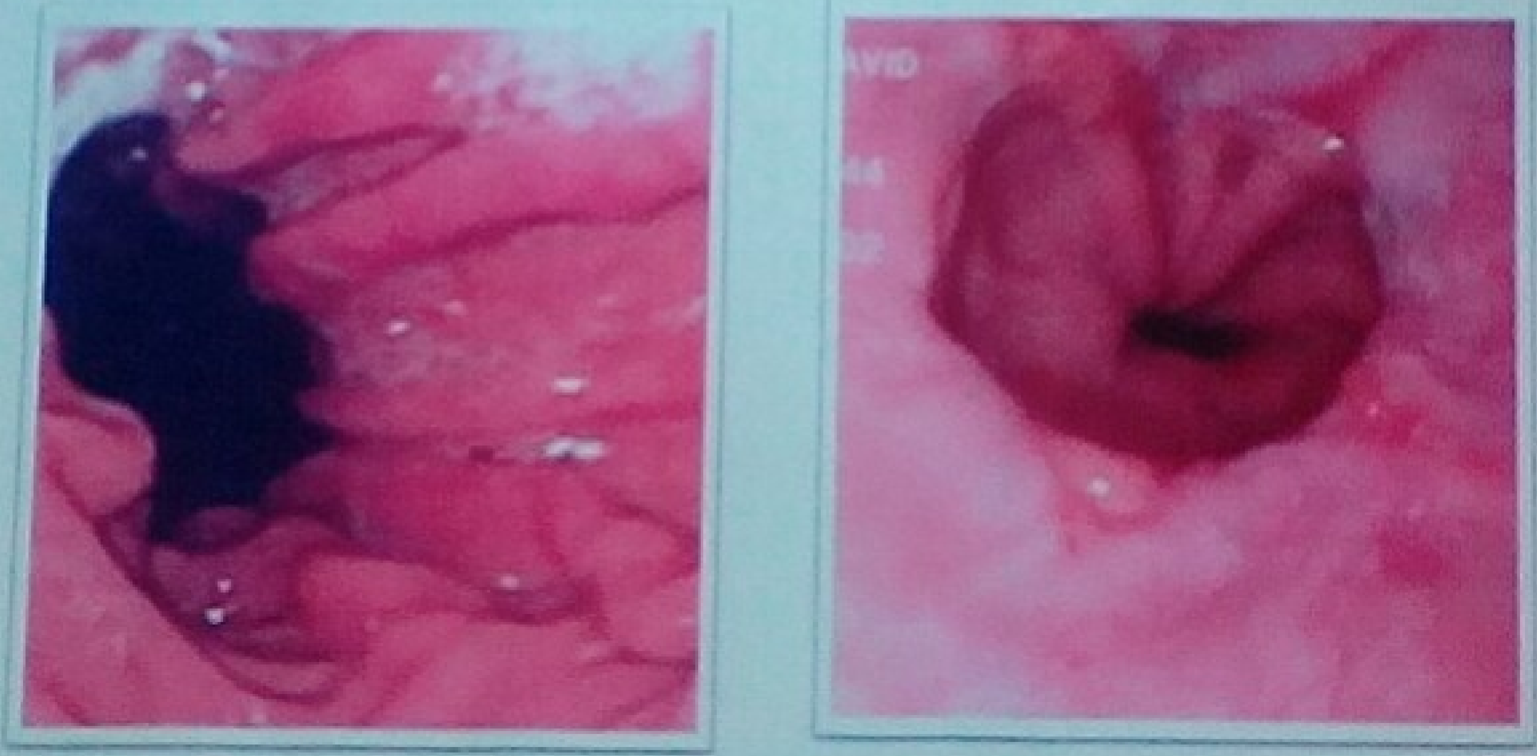
GERD esophagitis



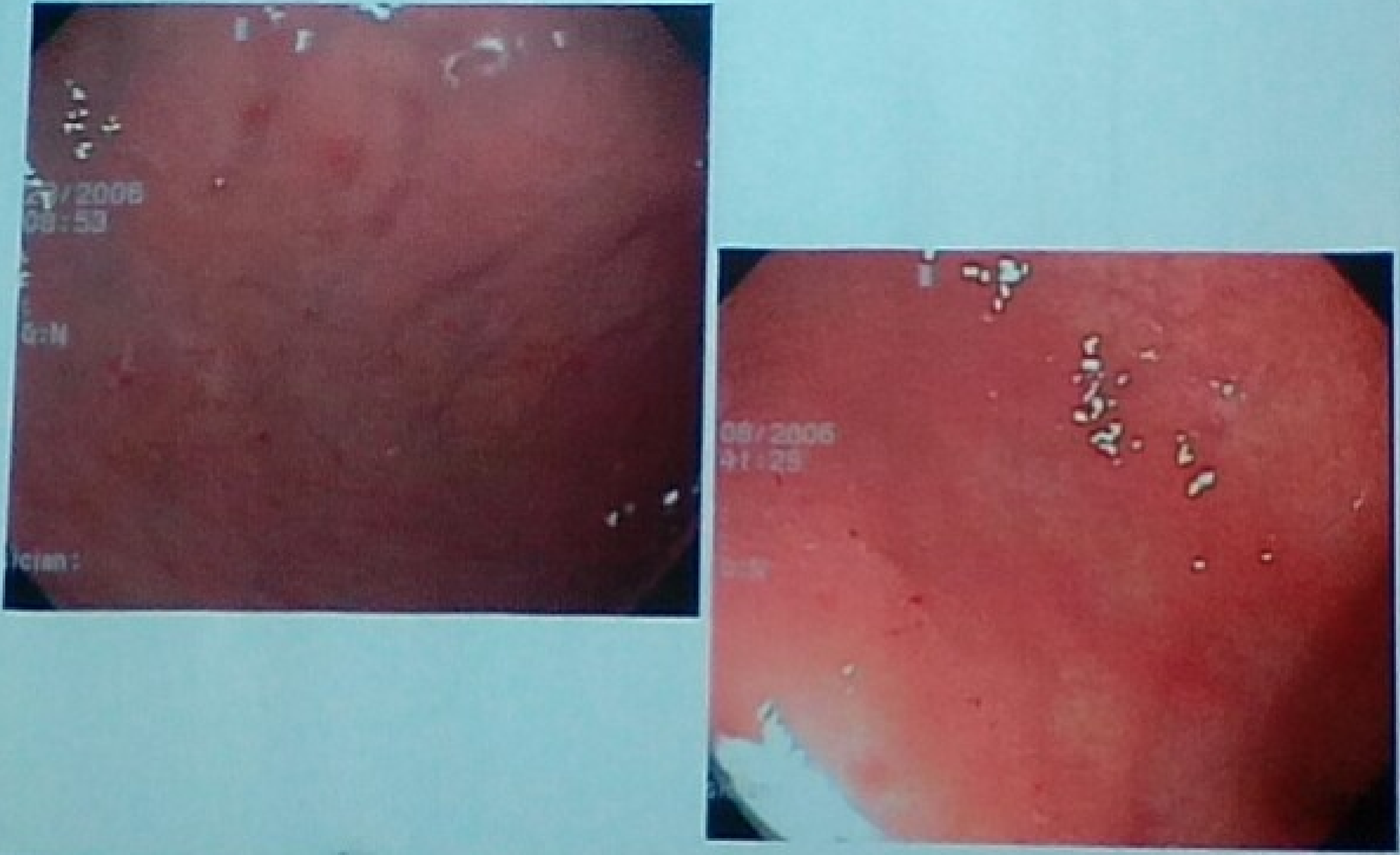
Nodular esophagus



Sliding HH



Gastritis



A 2 months old male, was delivered at term with birth weight 3,100 g presented with recurrent vomiting that started at age of 2 weeks. He received IV fluid therapy several times.

O/E, he looks lethargic, weight 3,250 g., dry tongue

B) What are items in history you want to ask??

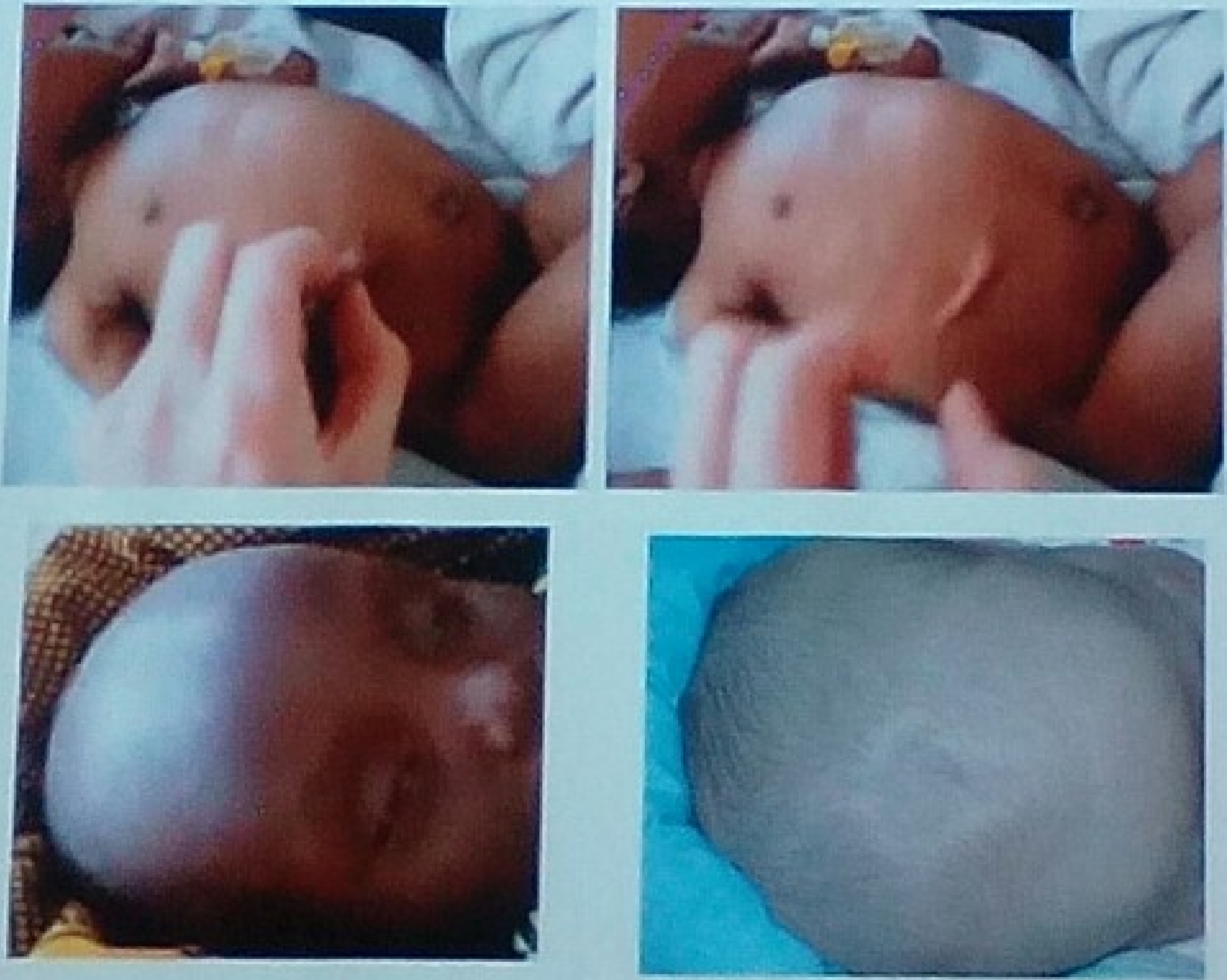
Important points in history

- Age of onset:
- Analysis of vomiting: bilious or non.
- Hematemesis
- Abdominal distension & absolute constipation
- Antenatal history: Oligohydramnios in obstructive uropahty, polyhydramnios in obstructive GI lesions.
- Dietetic history:

Important points in history

- Neurological: convulsions, disturbed LOC
- Urine output: polyurea in renal tubular defects & DKA
- FH of consanguinity, similar condition, sibling death

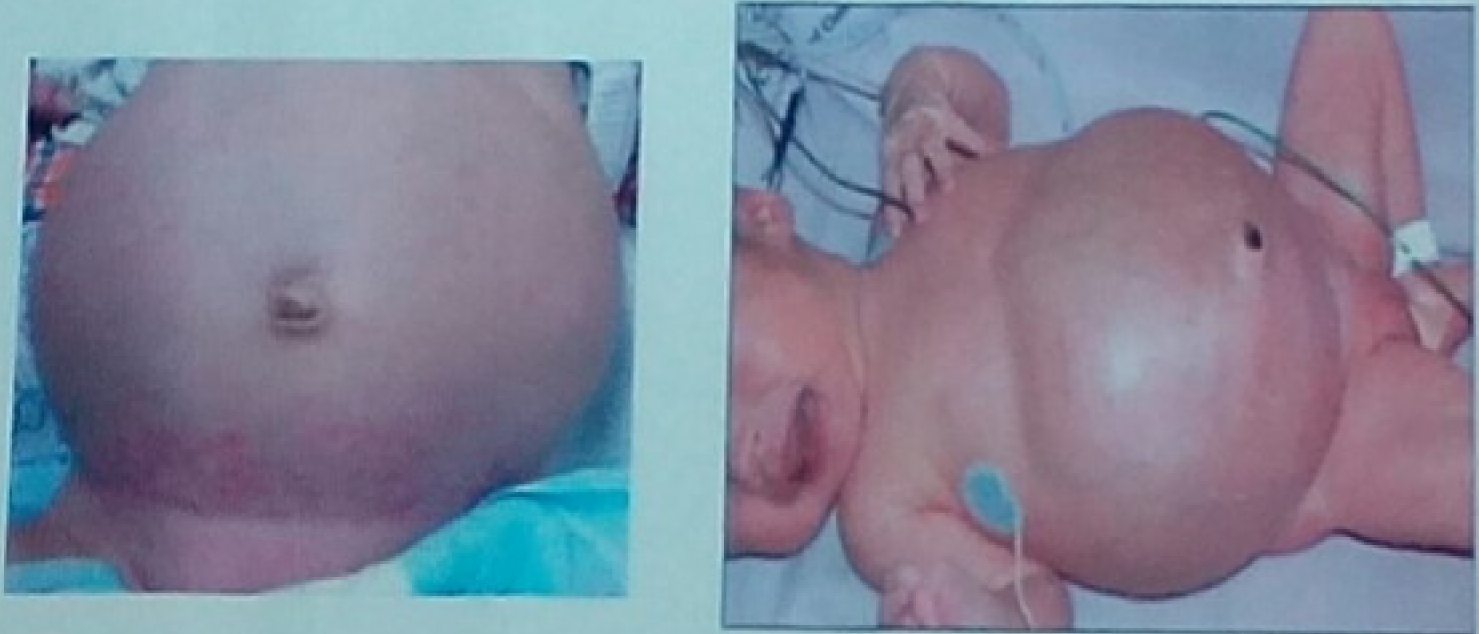
Signs of dehydration



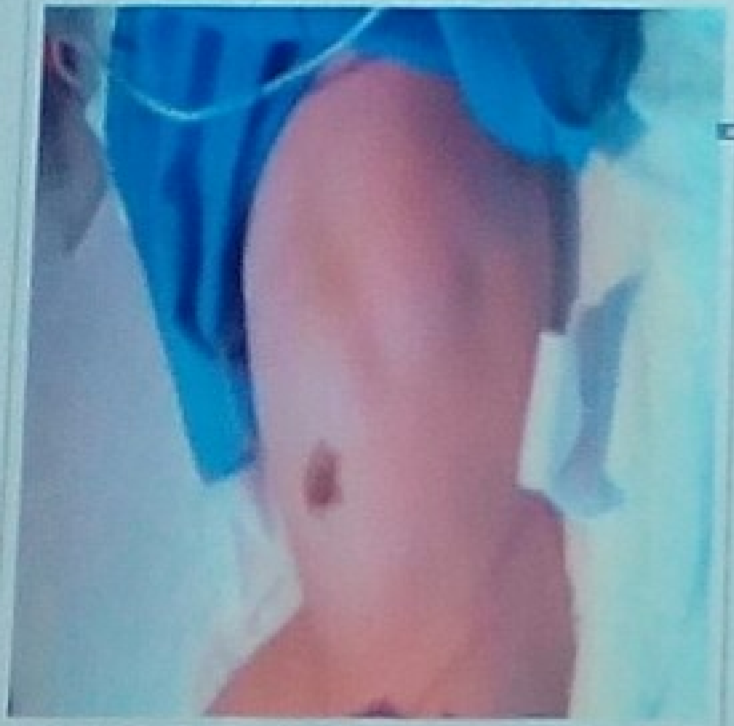
Local Abdominal examination

- Abdominal distension
- Look for **gastric peristalsis** (waves of muscle contraction across the abdomen passing from the left upper quadrant to the right lower quadrant).
- Pyloric mass: Firm, movable, approximately 2 cm in length, olive shaped, hard, best palpated from the left side, and located above and to the right of the umbilicus in the mid epigastrium beneath the liver edge.
- Organomegaly

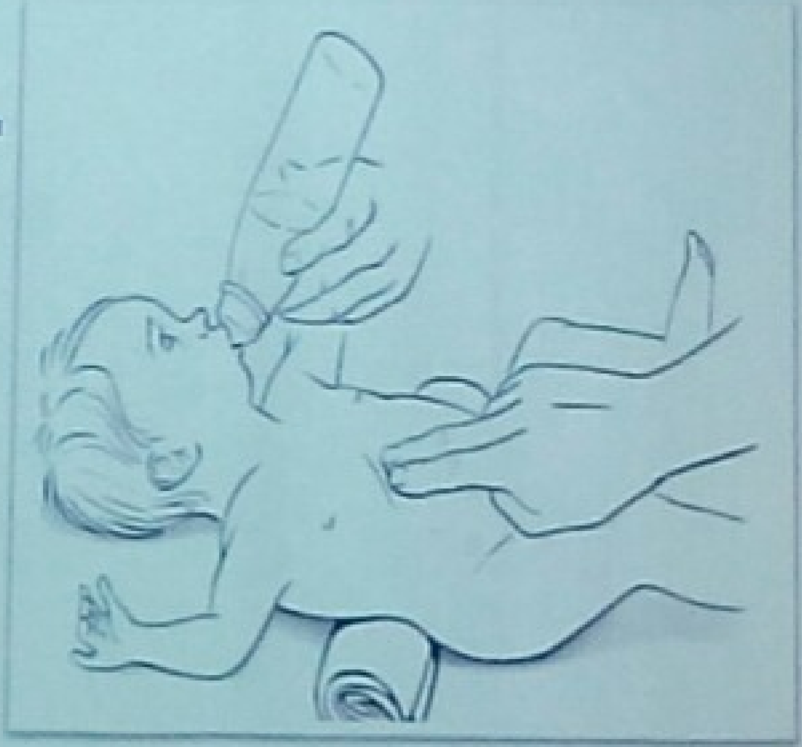
Abdominal distention



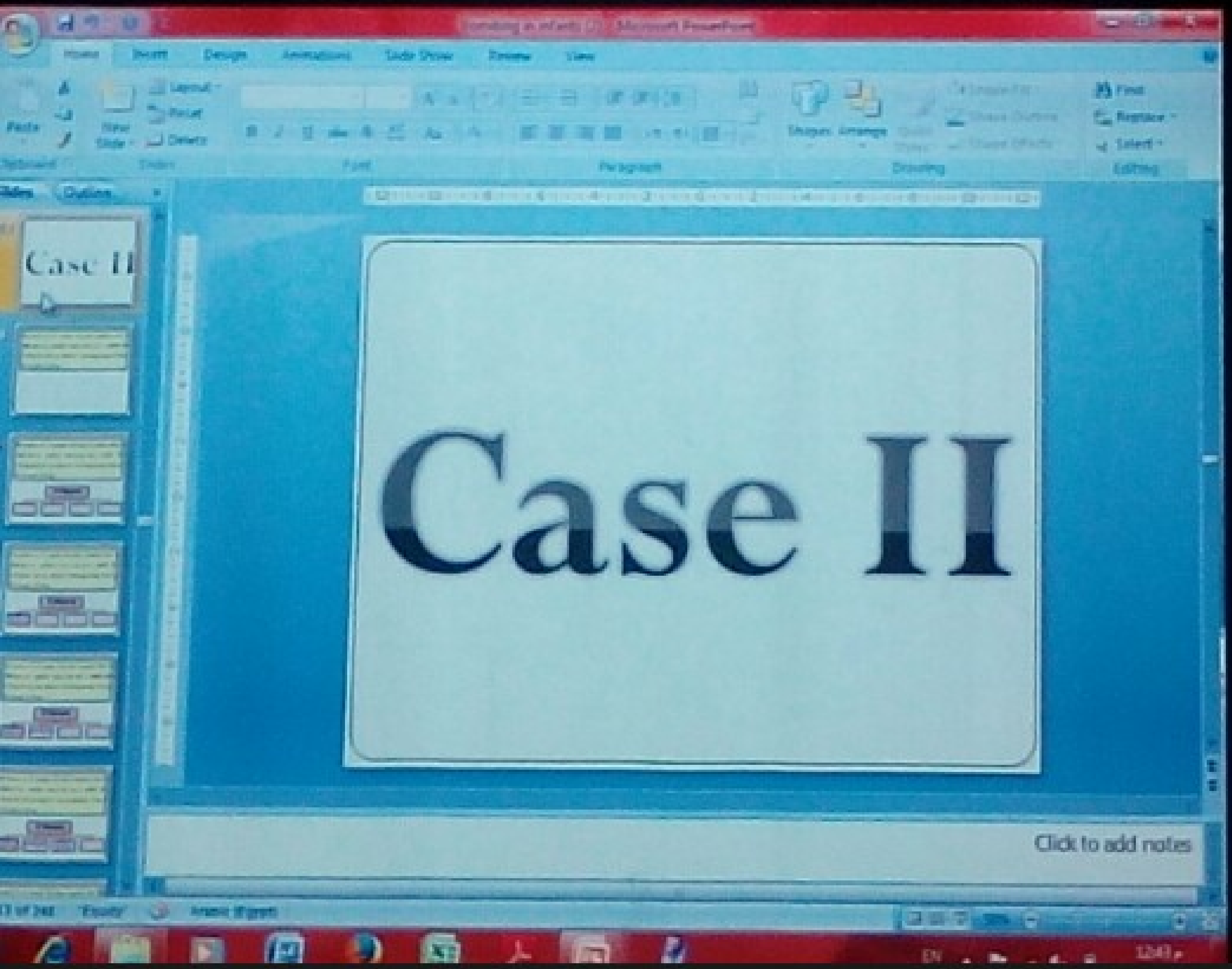
CHPS



Gastric peristaltic wave



Olive sign



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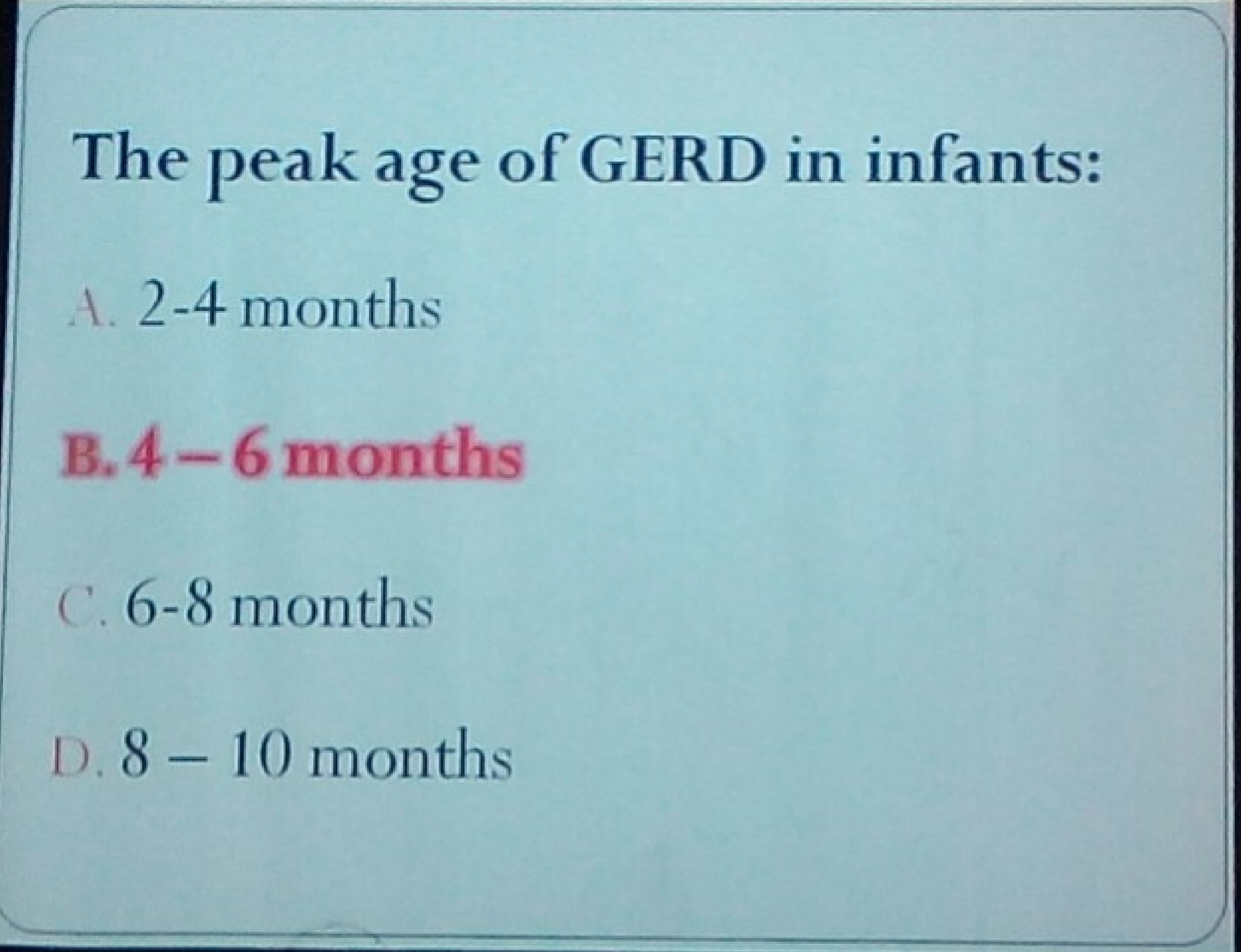
Animations
Slide Show
Review
View
Click to add notes

Case II



The peak age of GERD in infants:

A. 2-4 months

B. 4 – 6 months

C. 6-8 months

D. 8 – 10 months

The frequency of GERD at 4 months old:

A. 30%

B. 40%

C. 50%

D. 60%

The longest duration reported for use of PPI in children was

A. One year

B. 3 years

C. 5 years

D. 9 years

E. 11 years

Pediatrics, 2013;131:e1684–e1695

- Pediatric Gastroesophageal Reflux Clinical Practice Guidelines:
- Joint Recommendations of the North American Society for
- Pediatric Gastroenterology, Hepatology, and Nutrition
- (NASPGHAN) and the European Society for Pediatric
- Gastroenterology, Hepatology, and Nutrition (ESPGHAN)

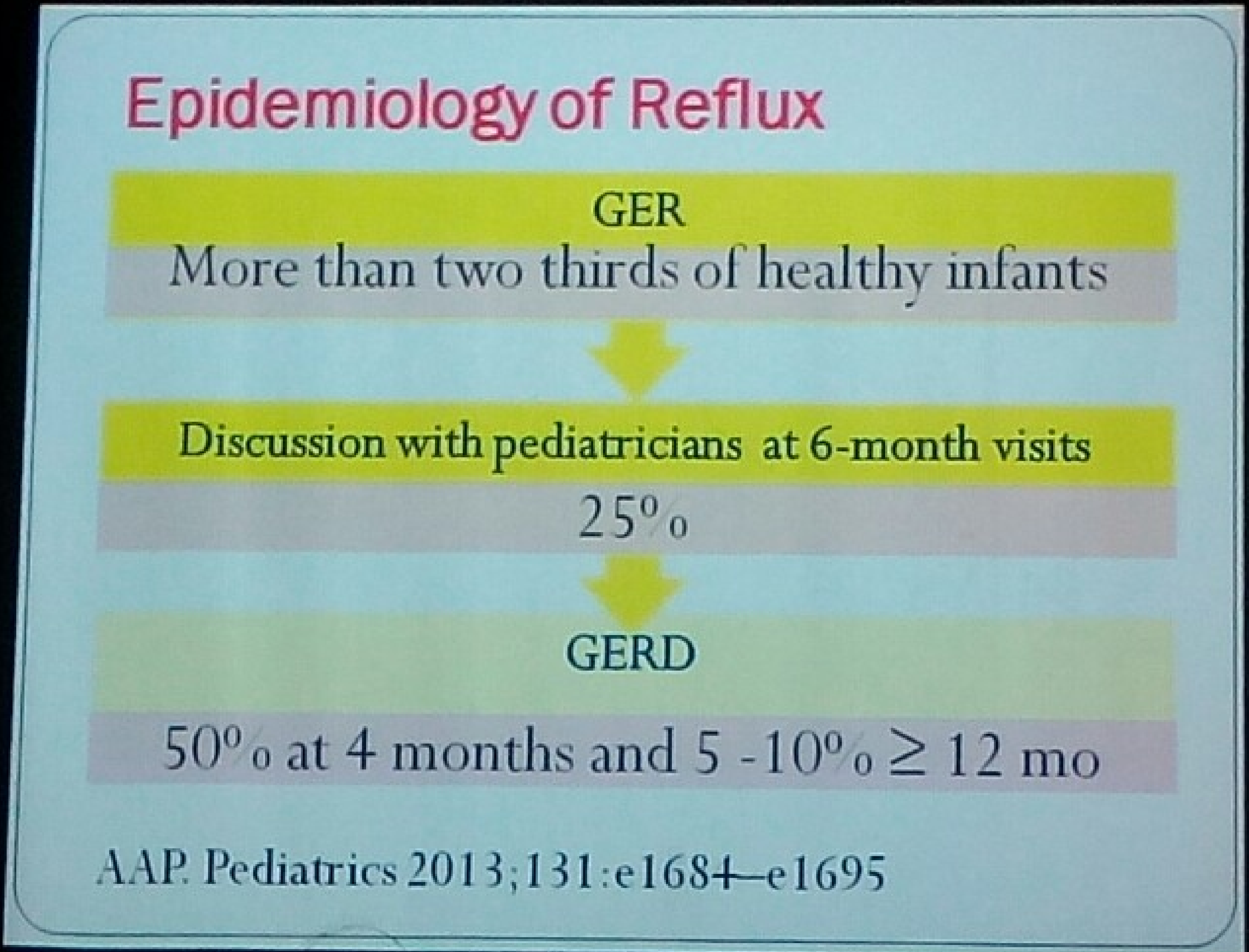
REFLUX

DEFINITIONS

GER, defined as the passage of gastric contents into the esophagus

Regurgitation: passive expulsion of gastric contents (Happy spitter)

GERD which includes **troublesome** symptoms or complications associated with GER



Epidemiology of Reflux

GER

More than two thirds of healthy infants

↓

Discussion with pediatricians at 6-month visits

25%

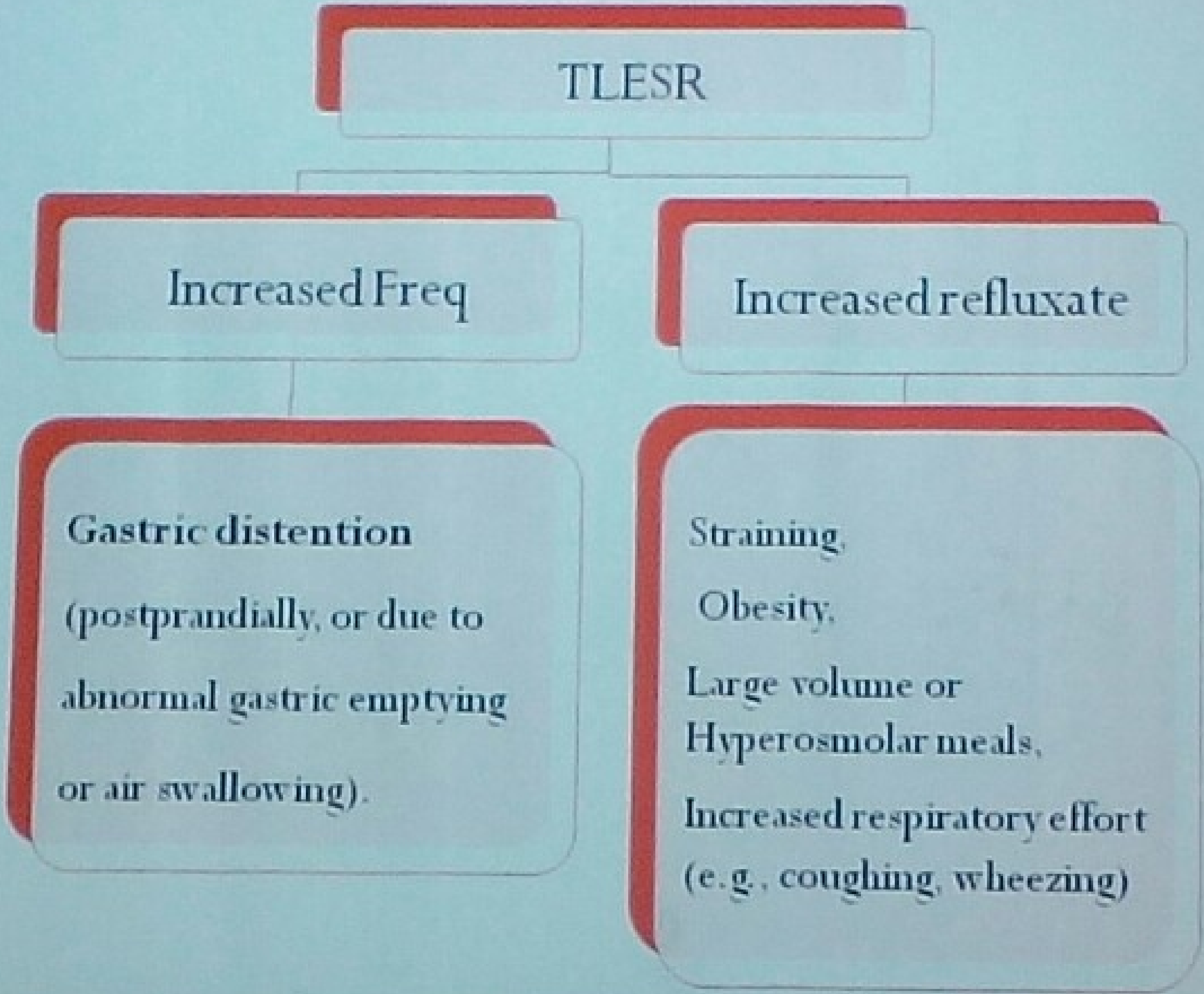
↓

GERD

50% at 4 months and 5 -10% ≥ 12 mo

AAP. Pediatrics 2013;131:e1684–e1695

ETIOLOGY &
PATHOPHYSIOLOGY



TLESR

Increased Freq

Gastric distention

(postprandially, or due to abnormal gastric emptying or air swallowing).

Increased refluxate

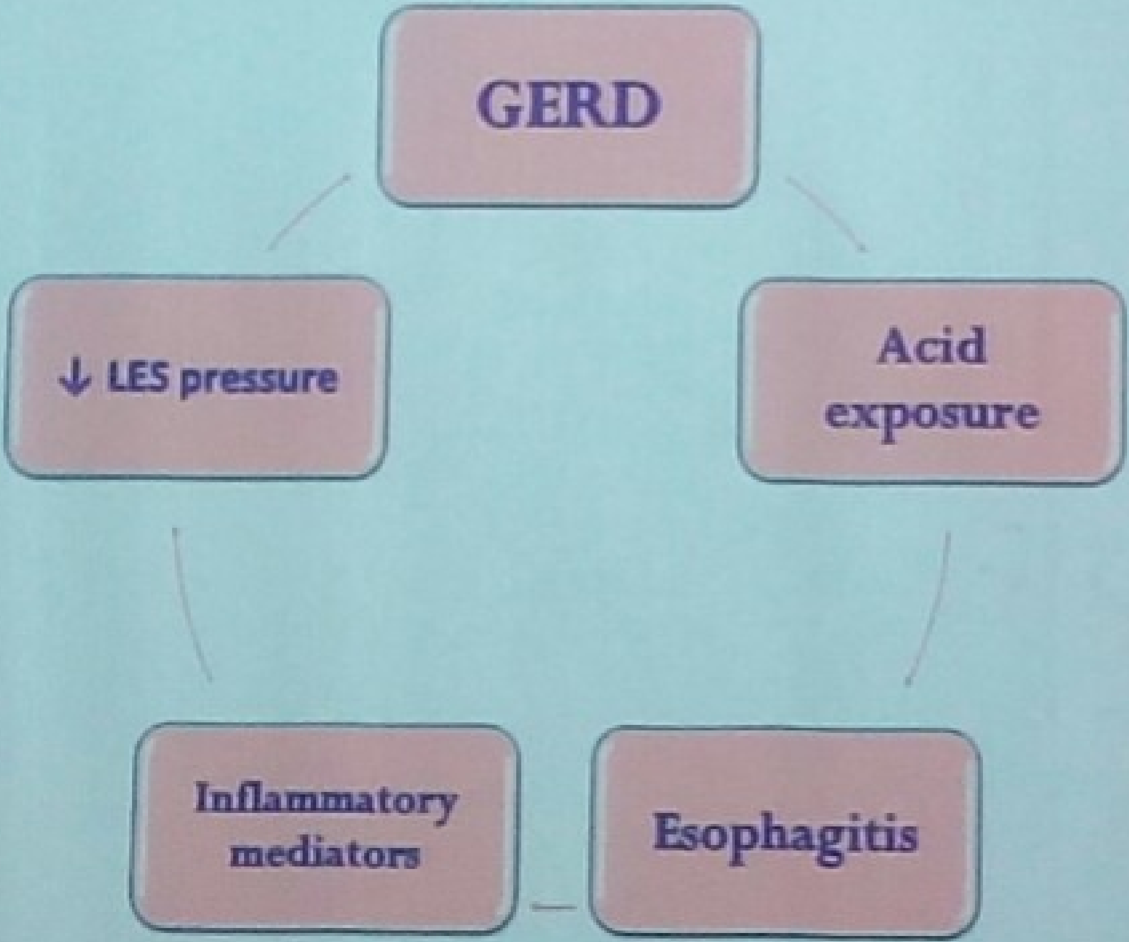
Straining,

Obesity,

Large volume or Hyperosmolar meals,

Increased respiratory effort (e.g., coughing, wheezing)

Patho-physiology



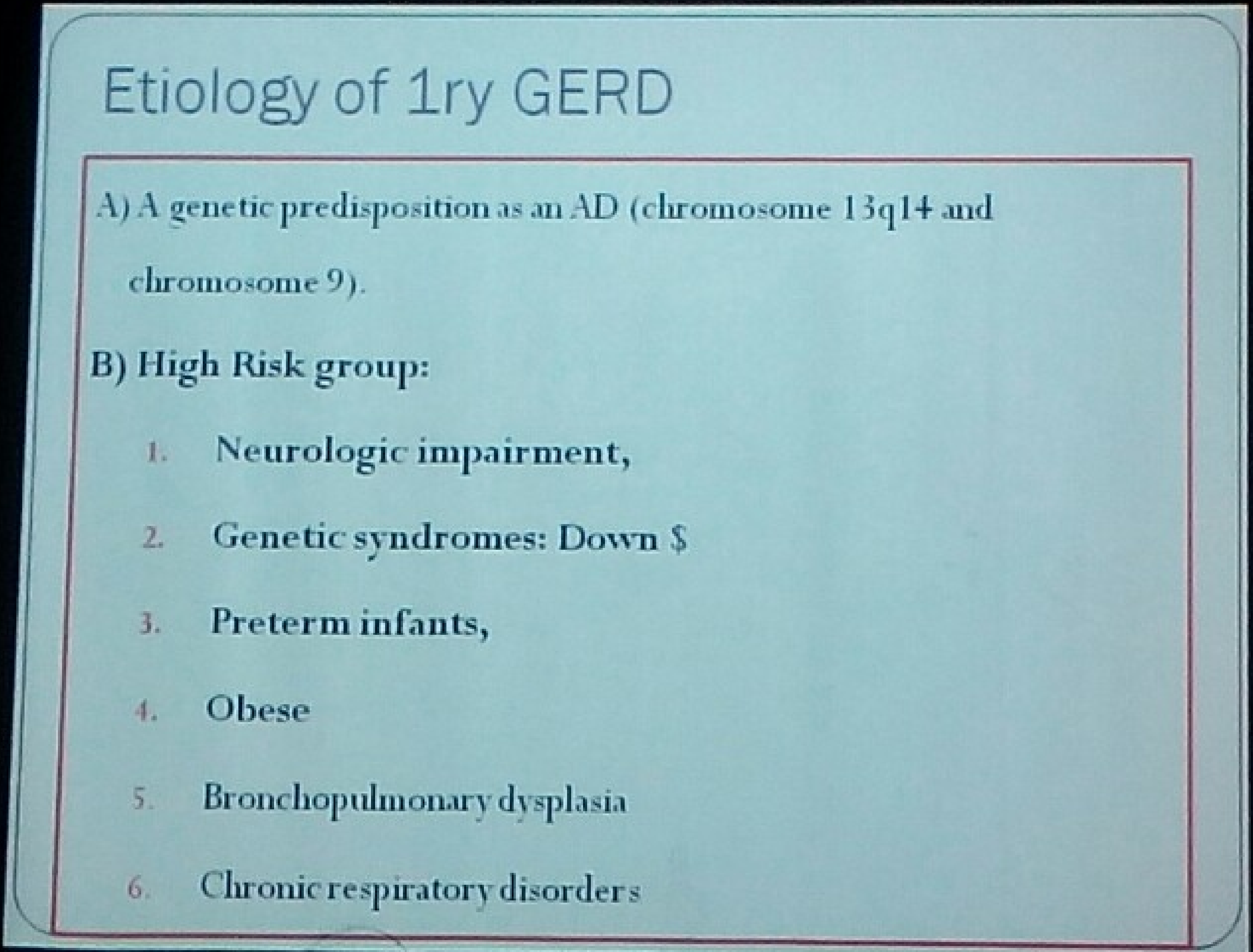
GERD

Acid exposure

Esophagitis

Inflammatory mediators

↓ LES pressure



Etiology of 1ry GERD

A) A genetic predisposition as an AD (chromosome 13q14 and chromosome 9).

B) High Risk group:

1. **Neurologic impairment,**
2. **Genetic syndromes: Down $**
3. **Preterm infants,**
4. **Obese**
5. Bronchopulmonary dysplasia
6. Chronic respiratory disorders

GERD

2ry

CMPA (25 - 50%)

Hiatus hernia

Gastric outlet obstruction

Tracheoesophageal fistula,

Eosinophilic esophagitis

1ry

Motility disorder with A genetic predisposition as an AD (chromosome 13q14 and chromosome 9.

Epidemiology of Reflux

GER

More than two thirds of healthy infants

Discussion with pediatricians at 6-month visits

25%

GERD

50% at 4 months and 5 -10% ≥ 12 mo

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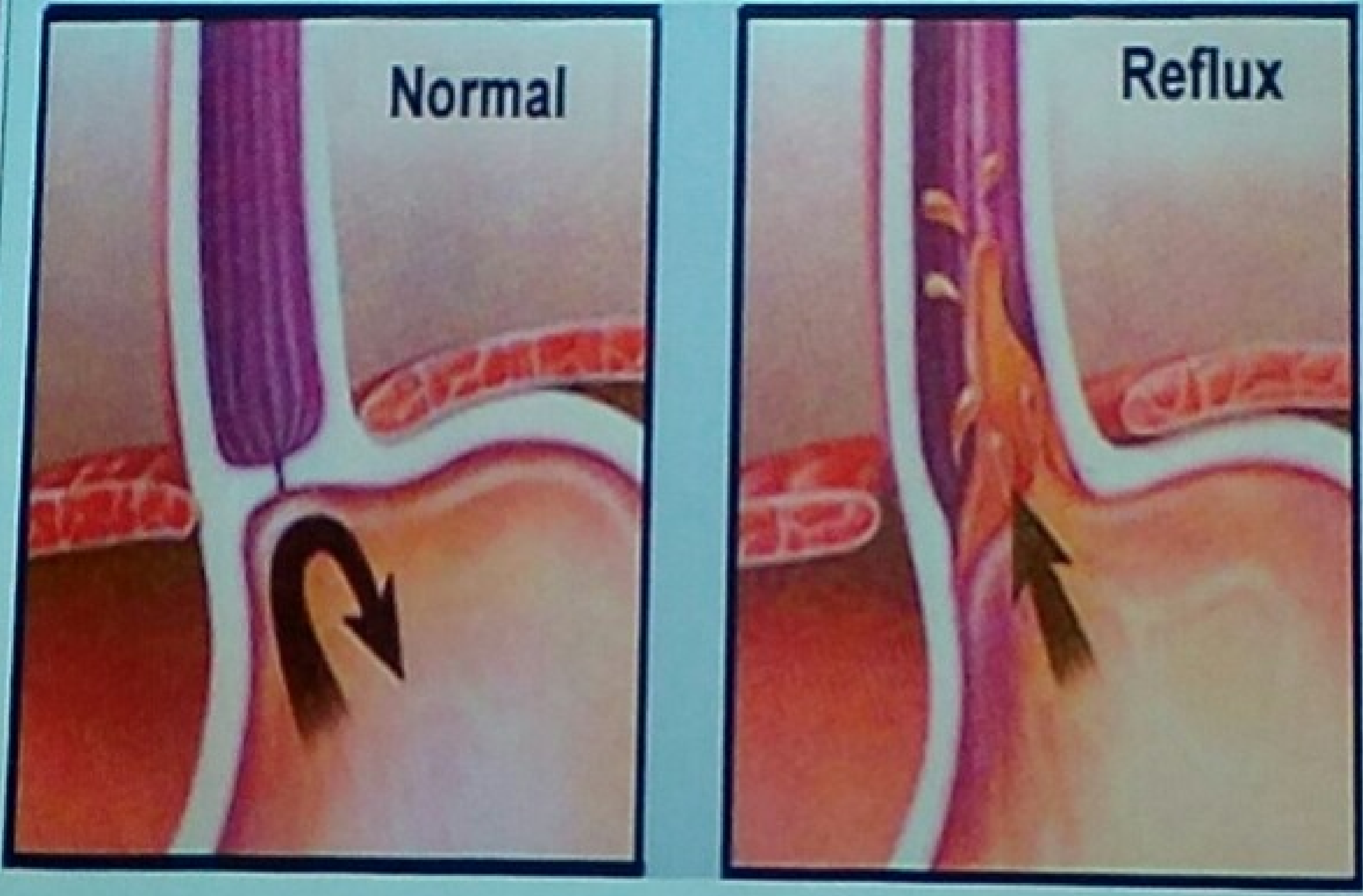
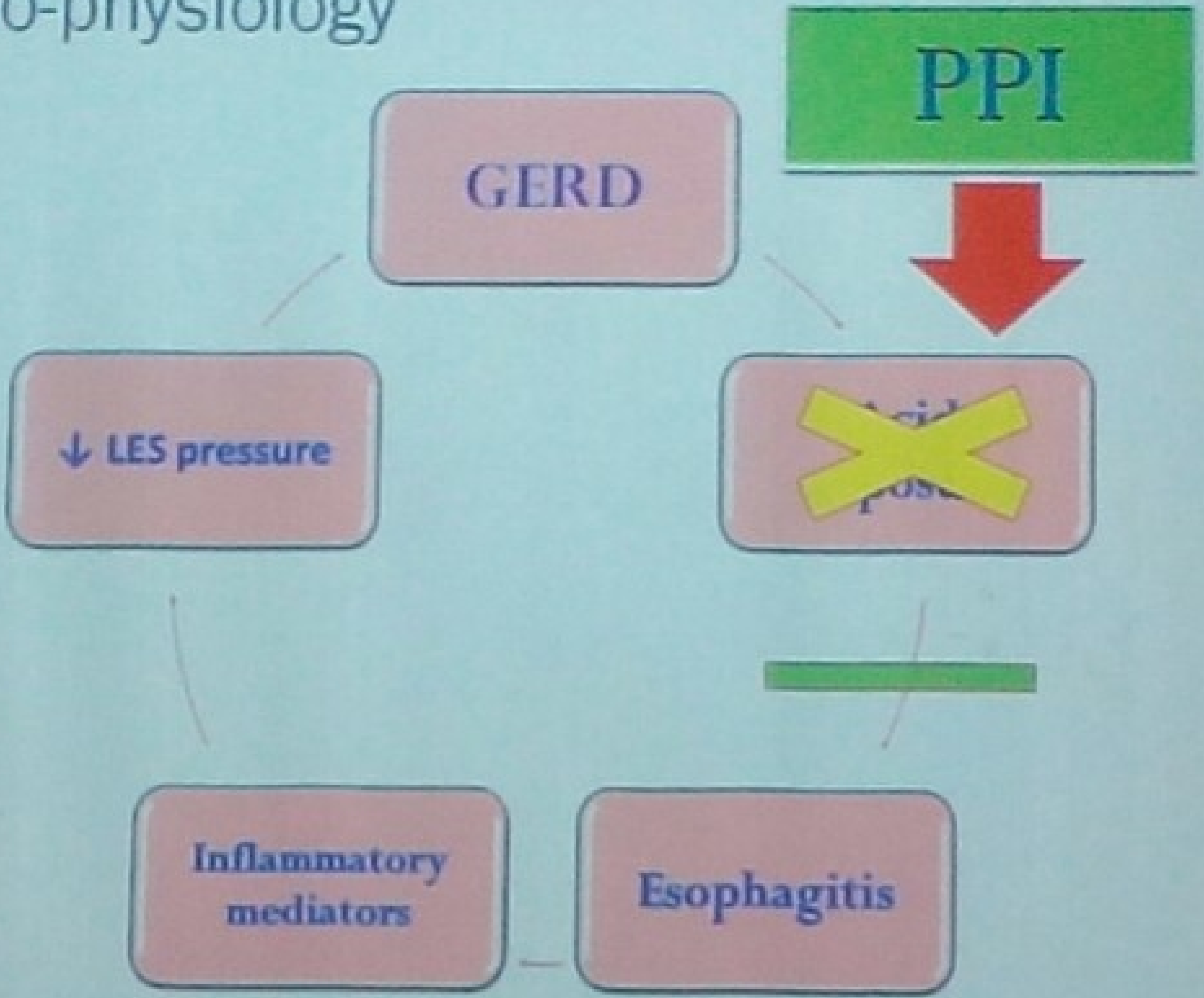

Normal
Reflux

Patho-physiology



PPI

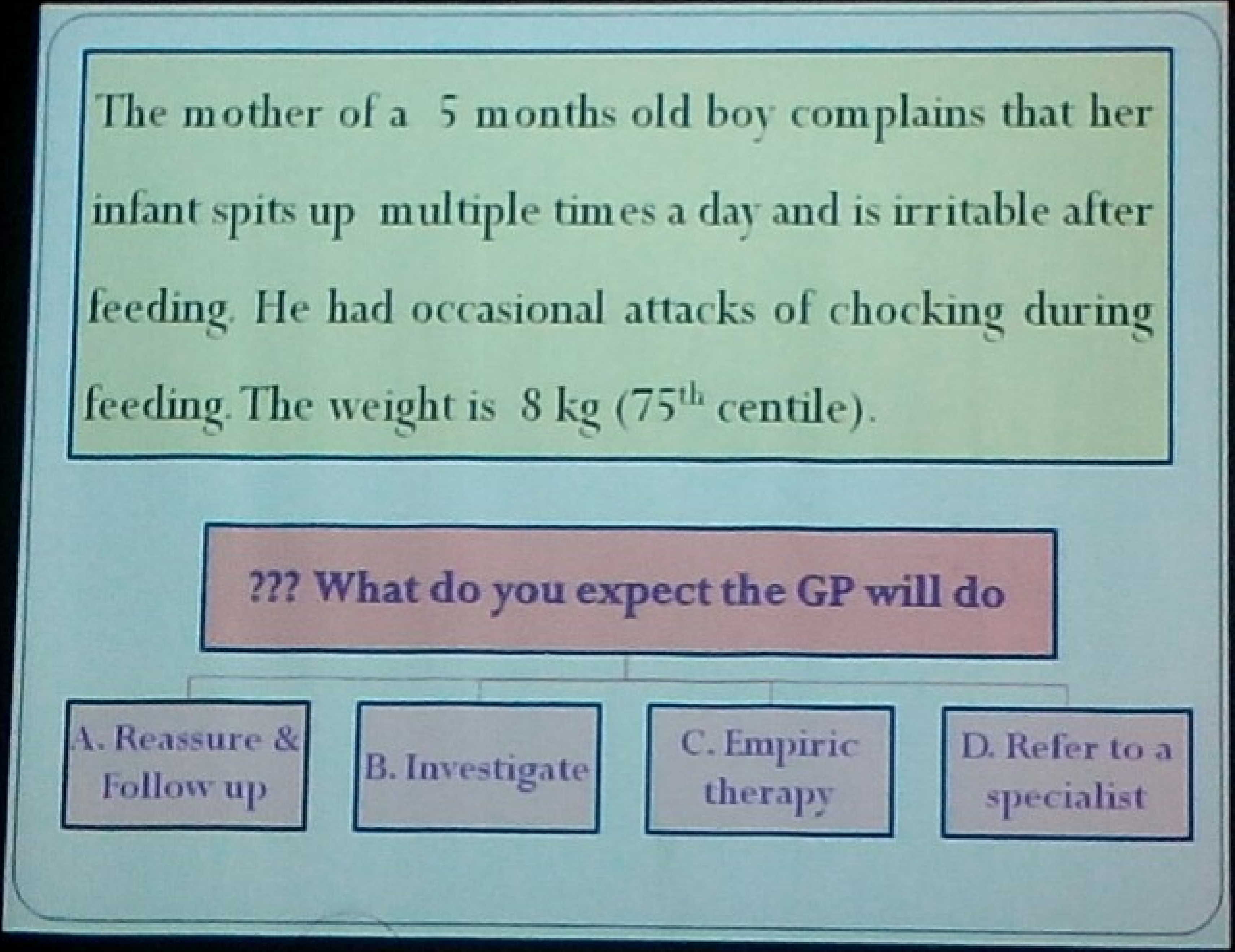
GERD

~~Acid exposure~~

Esophagitis

Inflammatory mediators

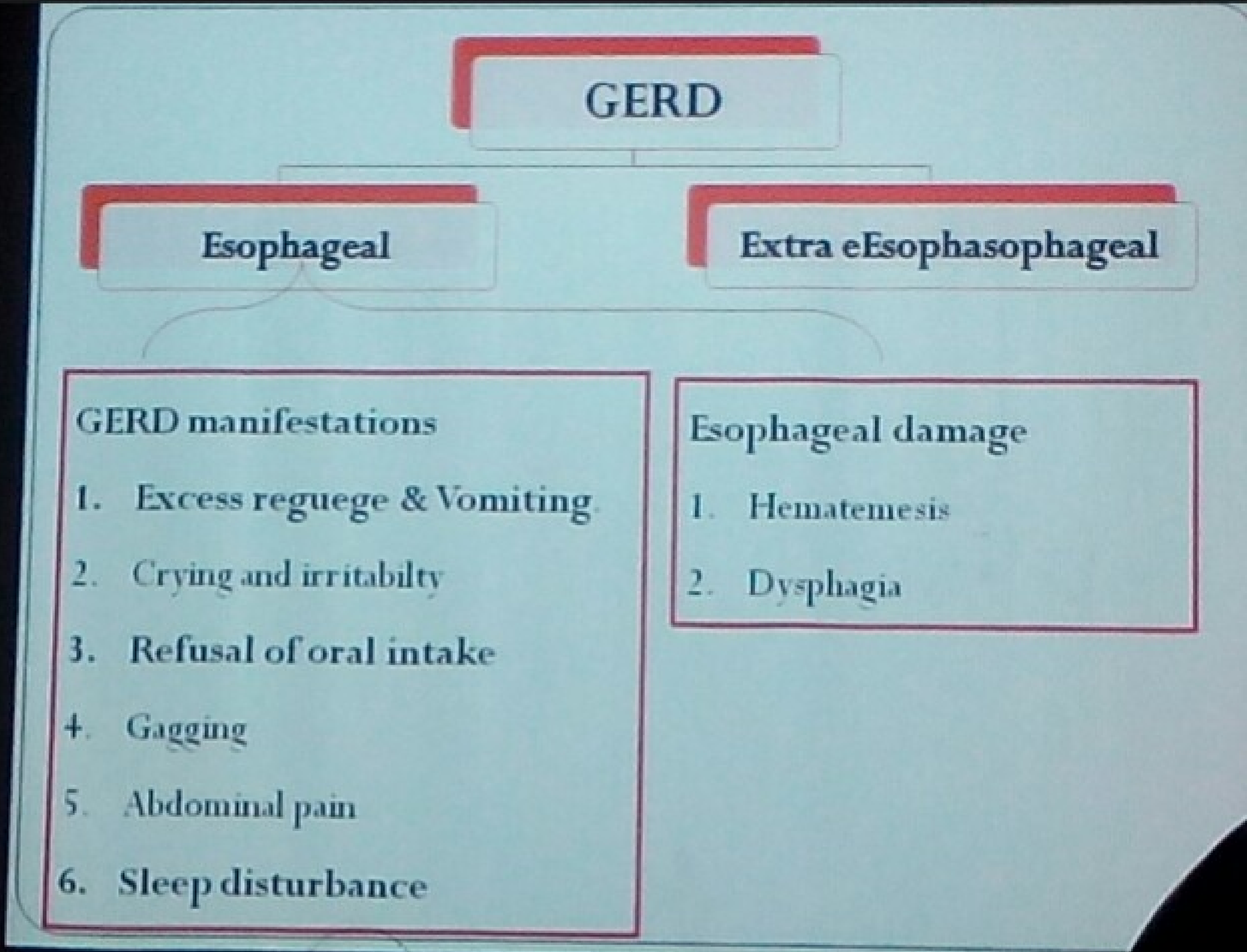
↓ LES pressure



The mother of a 5 months old boy complains that her infant spits up multiple times a day and is irritable after feeding. He had occasional attacks of chocking during feeding. The weight is 8 kg (75th centile).

??? What do you expect the GP will do

- A. Reassure & Follow up
- B. Investigate
- C. Empiric therapy
- D. Refer to a specialist



GERD

Esophageal

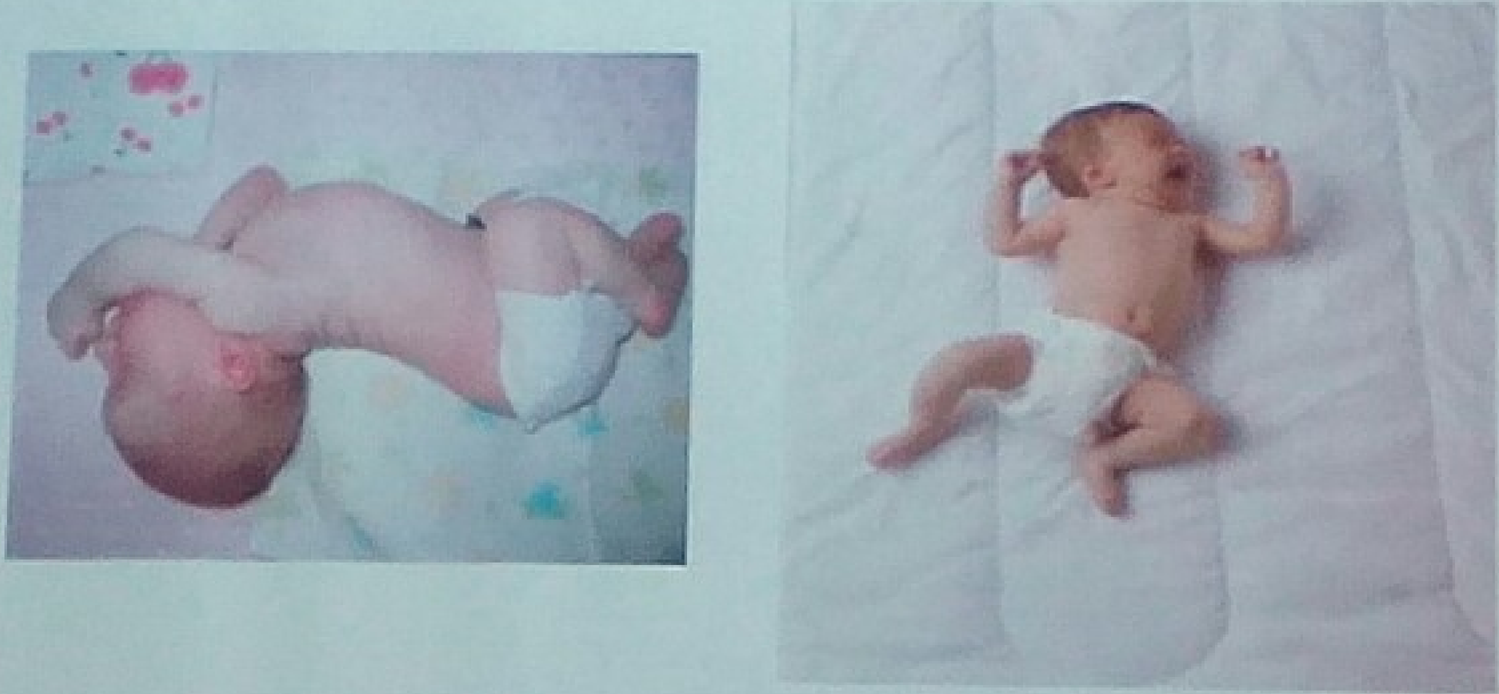
Extra eEsophasophageal

GERD manifestations

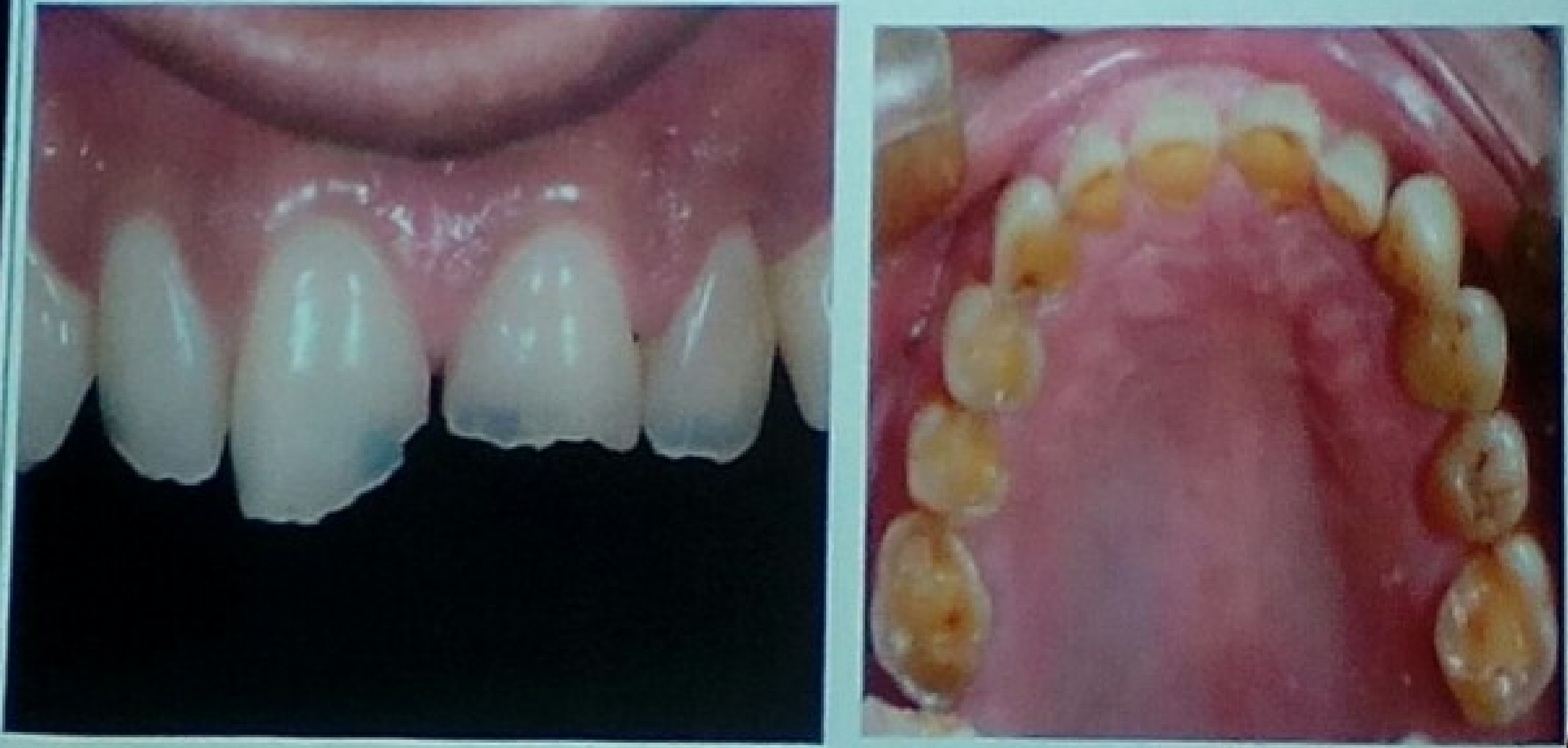
1. Excess reguege & Vomiting.
2. Crying and irritabilty
3. Refusal of oral intake
4. Gagging
5. Abdominal pain
6. Sleep disturbance

Esophageal damage

1. Hematemesis
2. Dysphagia



Dental erosions



The mother of a 5 months old boycomplains that her infant spits up multiple times a day and is irritable after feeding. He had occasional attacks of chocking during feeding. The weight is 8 kg (75th centile).

Based on the adequate weight gain, the family physician reassured the parents.

Weight gain with GERD

Generally, GERD causes troublesome symptoms **without necessarily interfering with growth**; however, children with clinically significant GERD or endoscopically diagnosed esophagitis may also develop an aversion to food, mostly because of a stimulus-response association of eating.

In infants and toddlers, there is no symptom or group of symptoms that can reliably diagnose GERD or predict treatment response. (B)

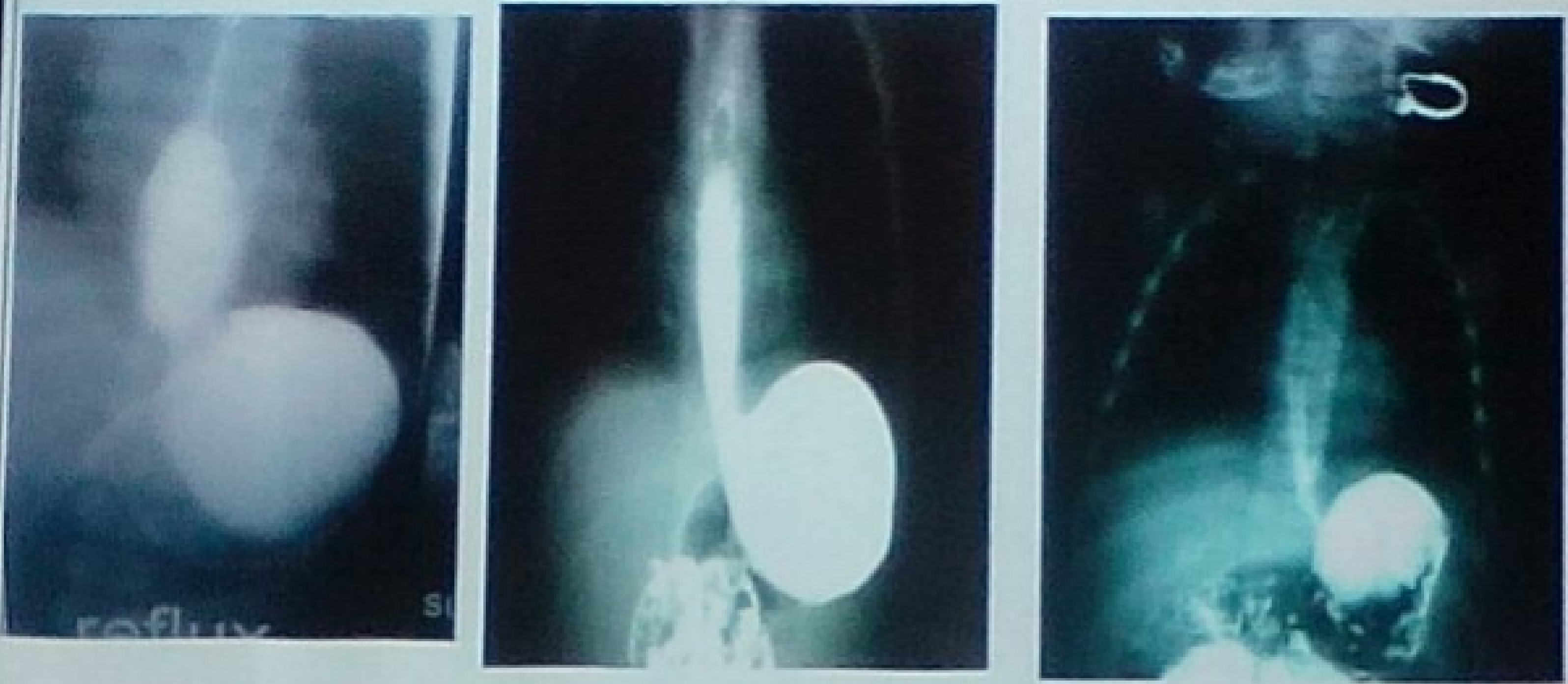
In older children and adolescents a history and physical examination are generally sufficient to reliably diagnose GERD and initiate management. (C)

NASPGHN. JPGN. 2009 Oct;49(4):498-547.

Barium studies

- Upper GI series
  - Barium swallow
  - Barium meal in Trendlenberg position
  - Barium meal
  - Barium meal with follow through

Upper GI series



Specificity of **50 %**

Sensitivity of **29%**

Barium studies

The upper gastrointestinal (GI) series is not useful for the diagnosis of GERD, but is useful for the diagnosis of anatomic abnormalities including hiatal hernia, and gastric outlet or intestinal obstruction . **(B)**

Performed in children with vomiting and dysphagia to evaluate for achalasia, esophageal strictures and stenosis.

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Esophageal pH metry

Esophageal pH monitoring is a valid and reliable measure of esophageal acid exposure only **(B)**

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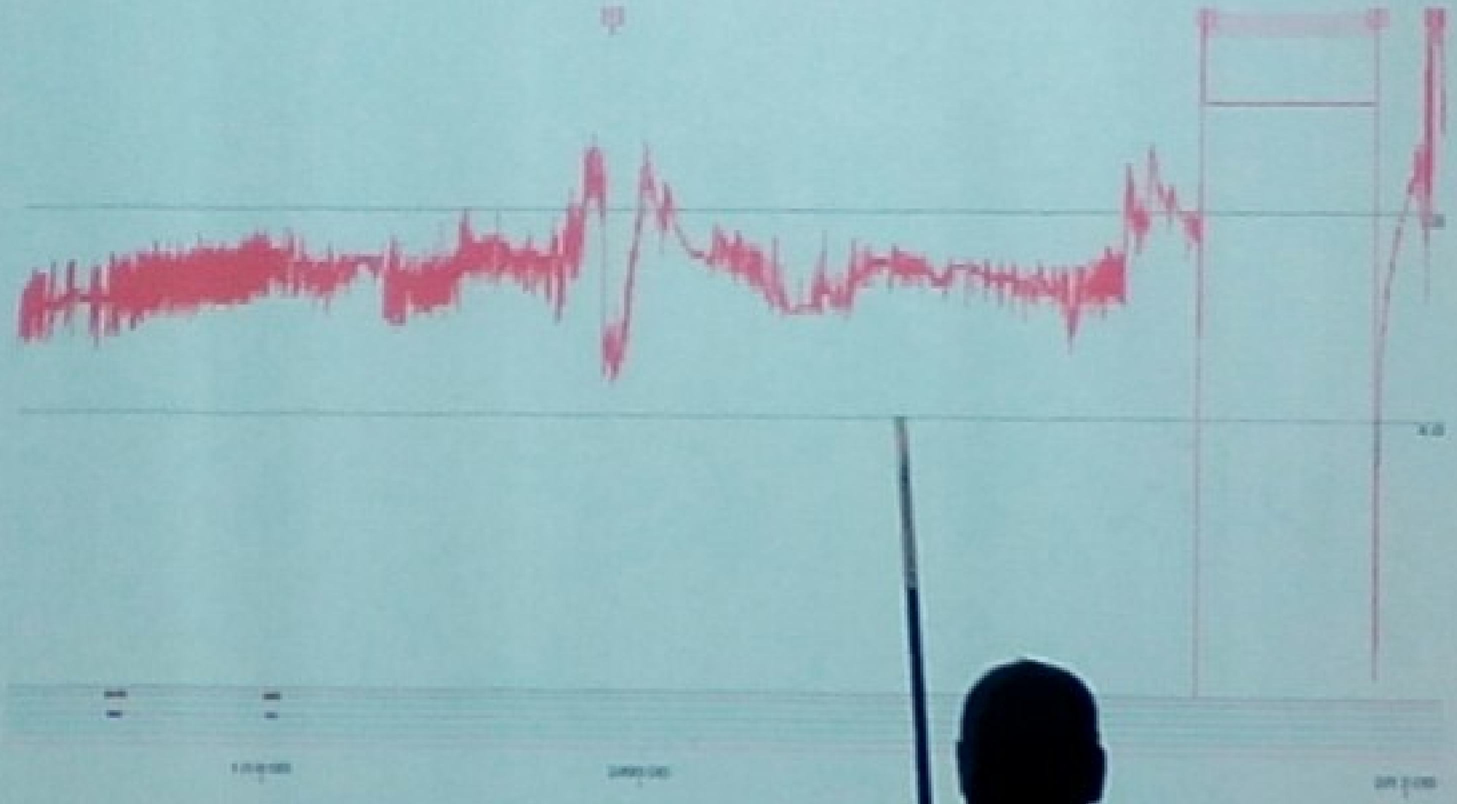
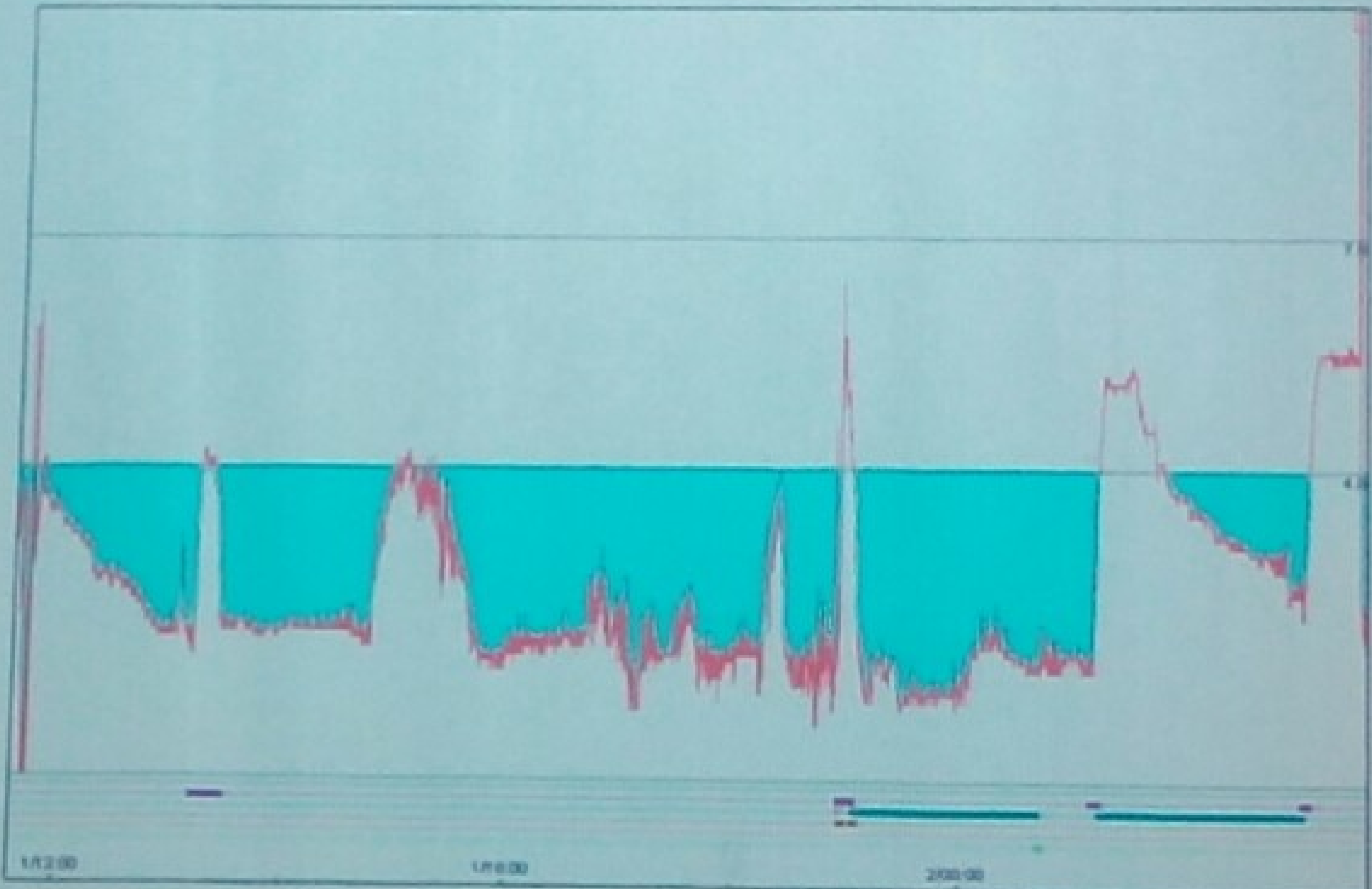
The most important indications for esophageal pH monitoring are :

1. Apneic episodes
2. Chronic cough,
3. Stridor,
4. Asthma.
5. Assess efficacy of acid suppressio[illegible] du[illegible] treatment

Normal Ph study



Severe acidic reflux



Limitation of pH metry

Evidence suggests poor reproducibility of pH testing,

In turn, esophageal pH monitoring is losing value as a primary modality for diagnosing managing pediatric GERD

Multiple intraluminal impedance (MII)

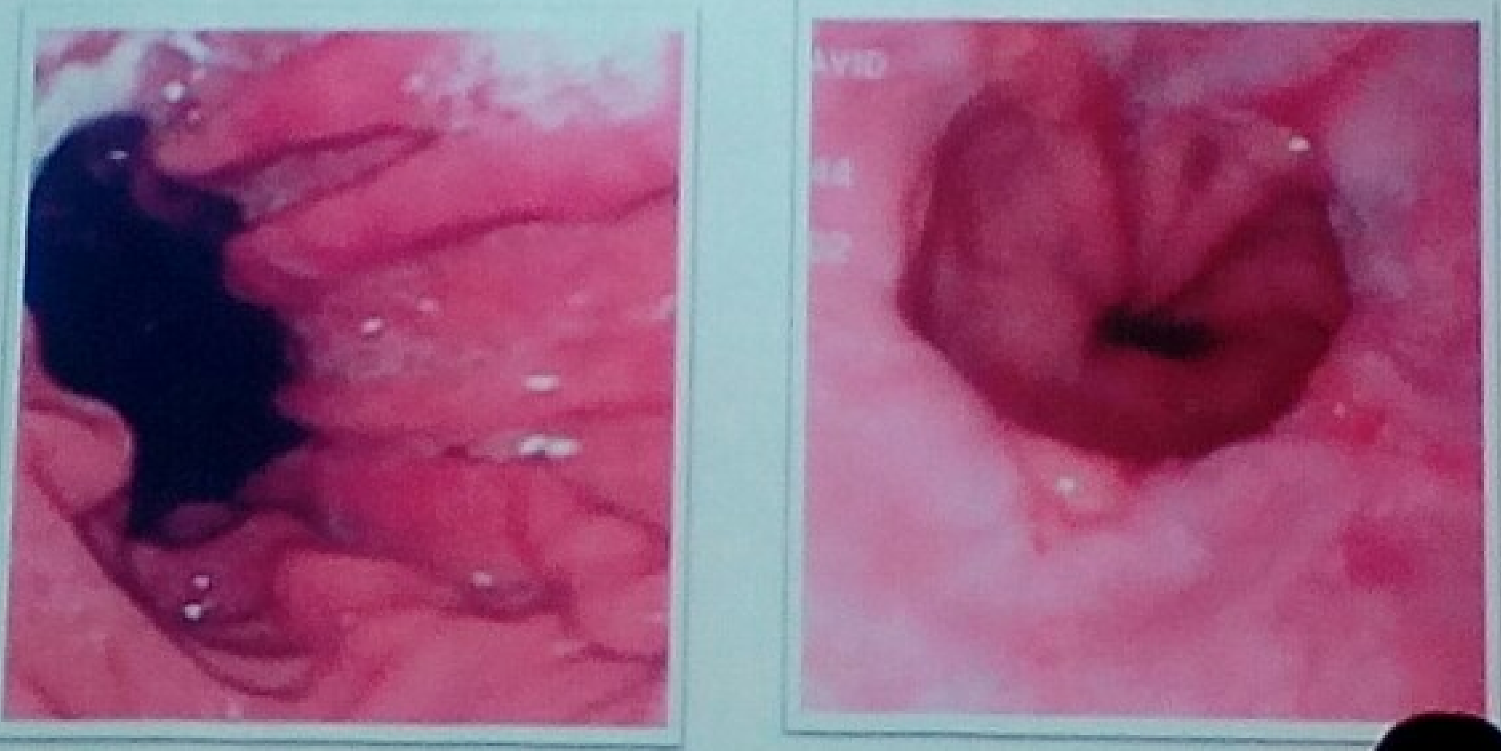
Esophageal impedance monitoring is considered the **most sensitive tool for assessing all types of GERD** (acidic, weakly acidic, non acidic and weakly alkaline)

4. Superimposed acid reflux (acid re-reflux),reflux while the pH is < 4 during an acid clearing interval (before the esophageal pH has recovered to > 4 after acid reflux);

Endoscopy in GERD

1. Hematemesis.
2. Poor weight gain,
3. Unexplained anemia or fecal occult blood,
4. Dysphagia
5. Failure of response to pharmacologic therapy

Sliding HH



Reflux Esophagitis (RE)

Endoscopically visible breaks in the distal esophageal mucosa are the most reliable evidence of reflux esophagitis (C)

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Limitations of EGD; NERD

However, the absence of endoscopic features of GERD does not exclude the diagnosis.

Despite pathological gastroesophageal reflux, the esophagus may be normal from the cricopharyngeus to the esophagogastric junction

Endoscopic biopsy

Endoscopic biopsy cannot determine whether esophagitis, if present, is due to reflux. (B)

Absence of histological changes does not rule out reflux disease. **(B)**

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The gold standard investigation for GERD is

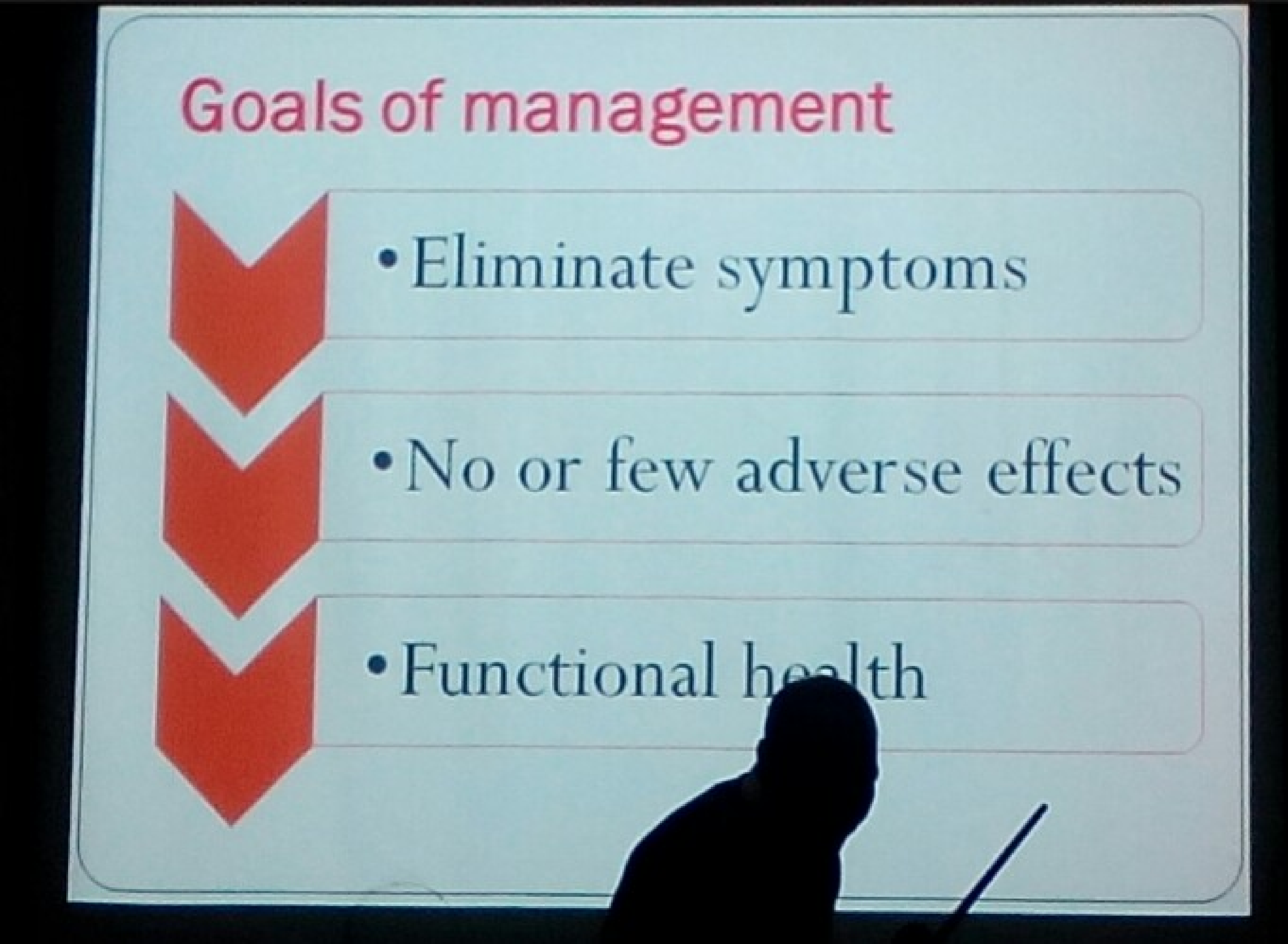
A. Barium study

B. Esophageal impedence with pH metry

C. Esophageal manometry

D. Endoscopy & biopsy

E. None



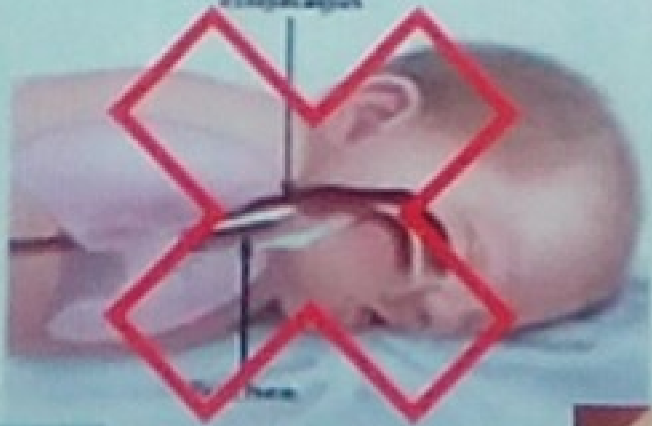
Goals of management

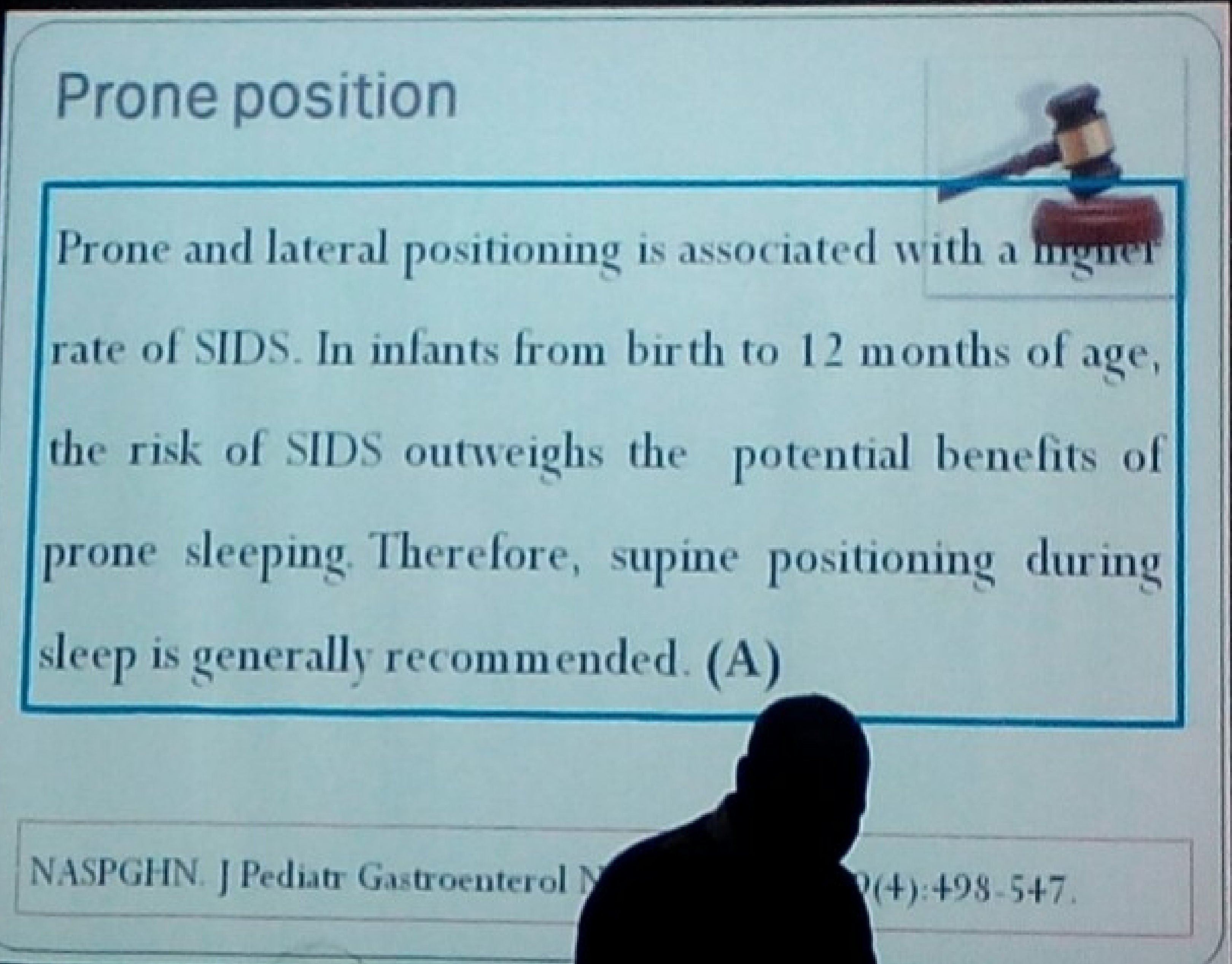
- Eliminate symptoms
- No or few adverse effects
- Functional health

Management; Conservative

1. Positioning
2. Dietary changes
3. Weight reduction for obese patients
4. Elimination of smoke exposure

fant positioning





Prone position

Prone and lateral positioning is associated with a higher rate of SIDS. In infants from birth to 12 months of age, the risk of SIDS outweighs the potential benefits of prone sleeping. Therefore, supine positioning during sleep is generally recommended. **(A)**

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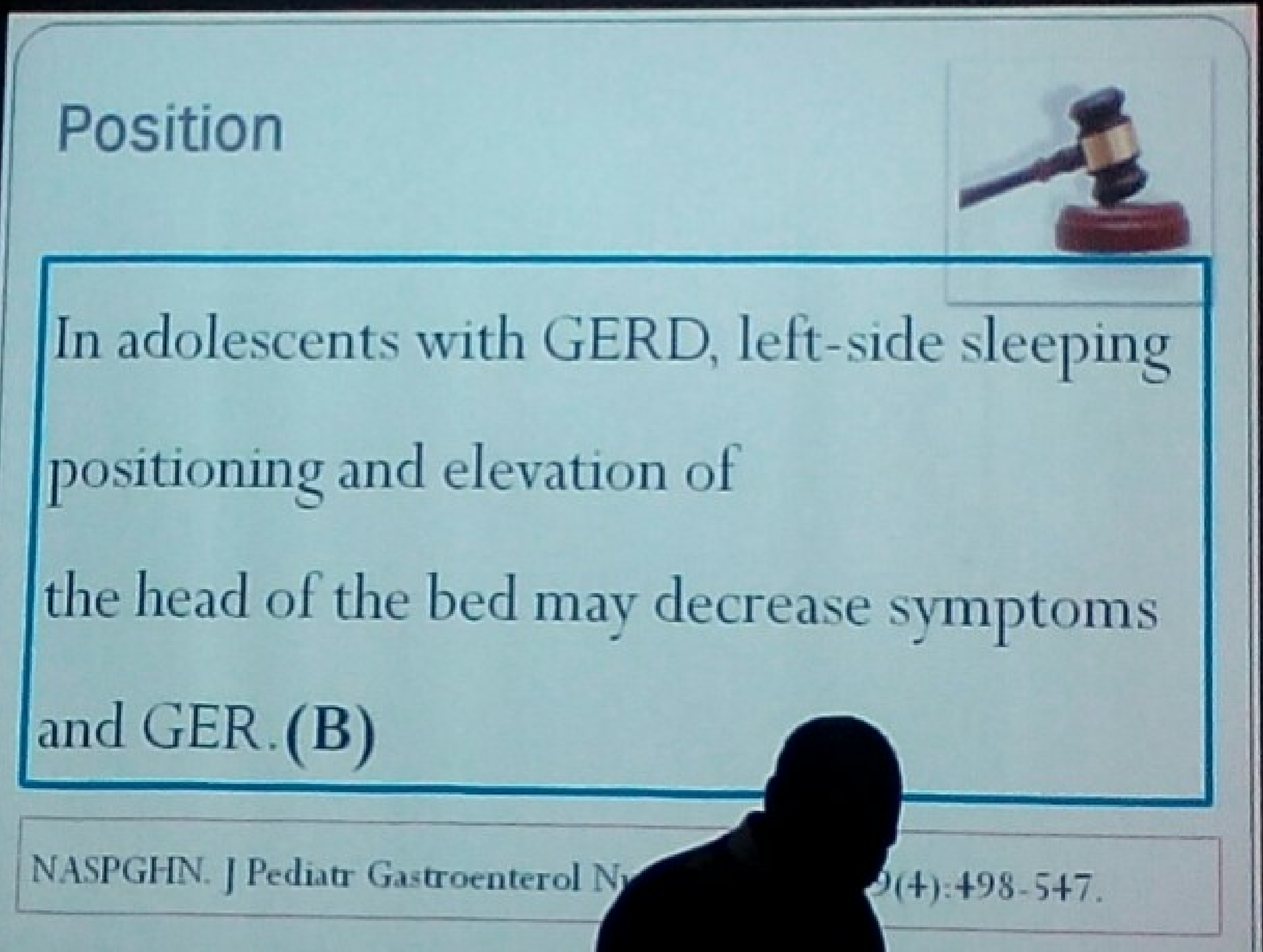

Position

In adolescents with GERD, left-side sleeping positioning and elevation of the head of the bed may decrease symptoms and GER. **(B)**

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Dietary changes

a 2- to 4-week trial of a maternal exclusion diet that restricts at least milk and egg is recommended in breastfeeding infants with GERD symptoms

There is evidence to support a trial of an extensively hydrolyzed protein formula for a 2- to 4-week trial in formula-fed infants with vomiting. (B)

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Dietary changes

Thickening of formula results in decreased visible reflux (regurgitation) . (A)

Thickened feedings appear to decrease observed regurgitation rather than the actual number of reflux episodes

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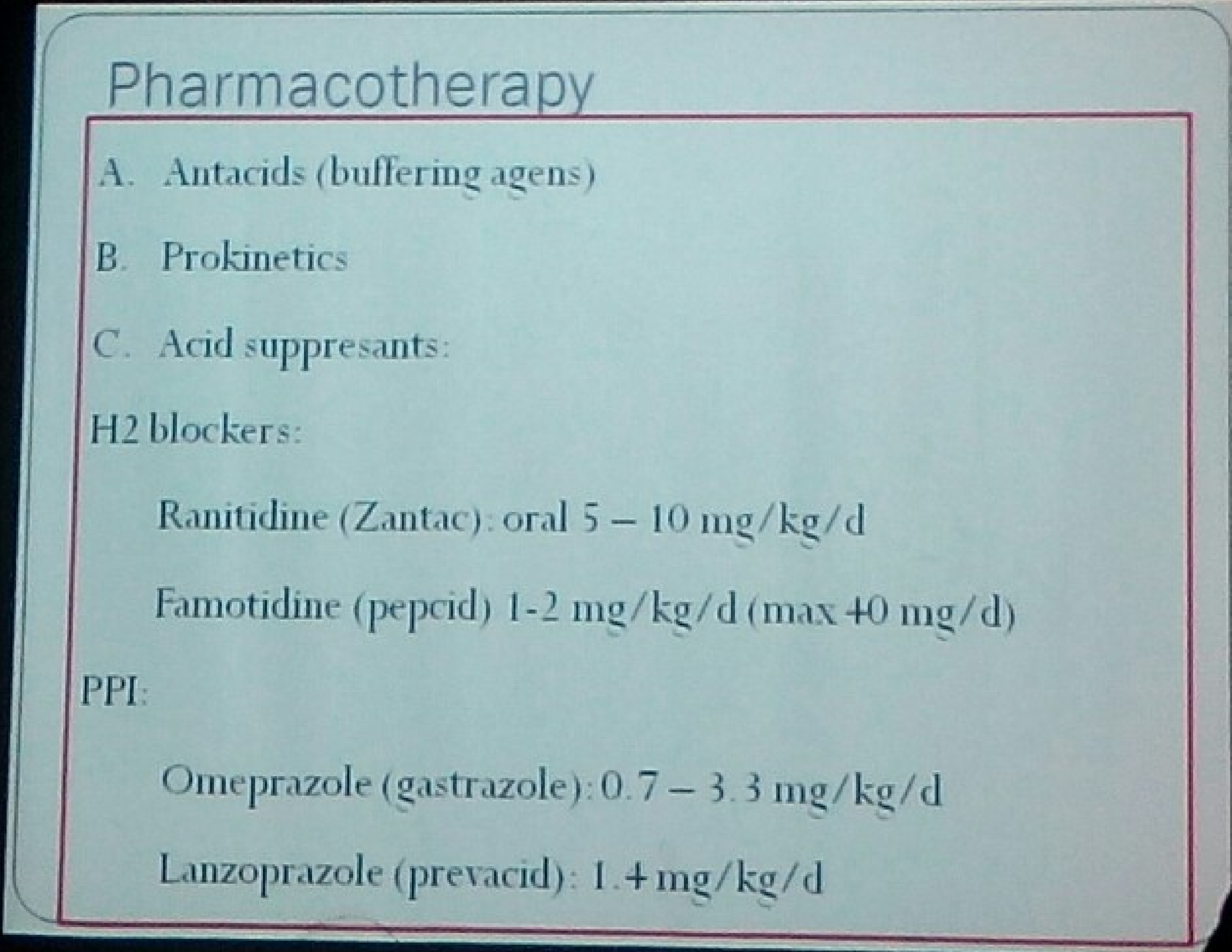


Pharmacotherapy

A. Antacids (buffering agens)

B. Prokinetics

C. Acid suppresants:

H2 blockers:

Ranitidine (Zantac): oral 5 – 10 mg/kg/d

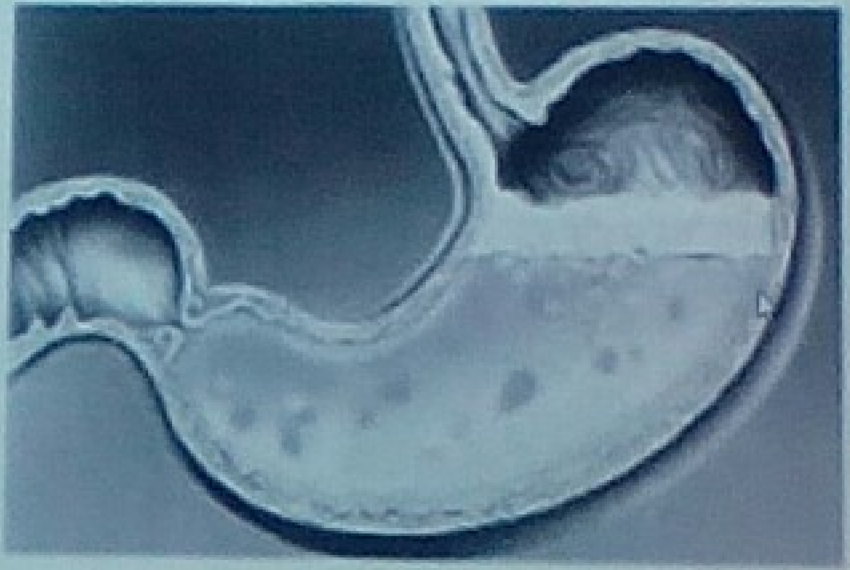
Famotidine (pepcid) 1-2 mg/kg/d (max 40 mg/d)

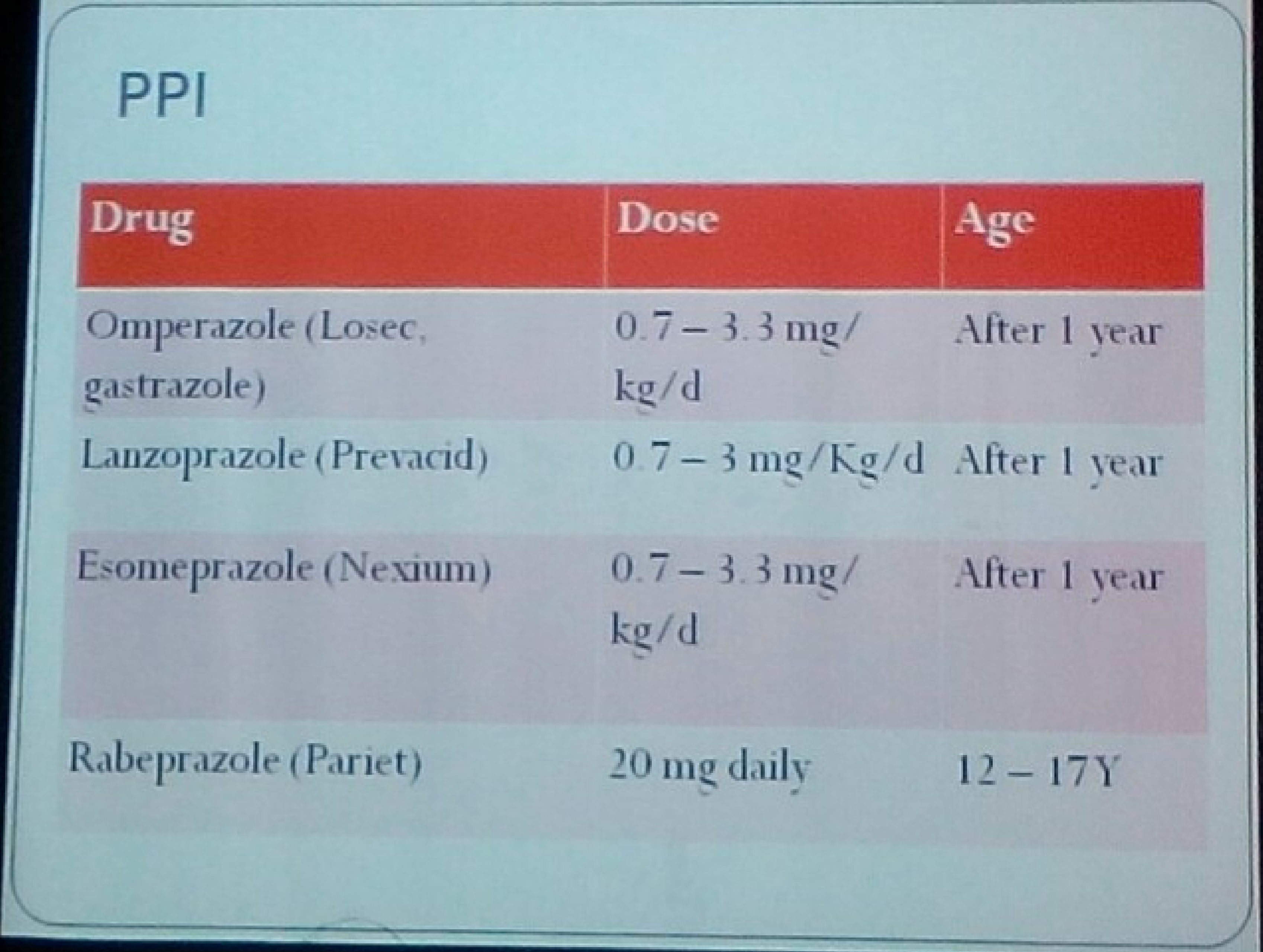
PPI:

Omeprazole (gastrazole): 0.7 – 3.3 mg/kg/d

Lanzoprazole (prevacid): 1.4 mg/kg/d

Buffering agents





PPI

Drug	Dose	Age
Omperazole (Losec, gastrazole)	0.7 – 3.3 mg/ kg/d	After 1 year
Lanzoprazole (Prevacid)	0.7 – 3 mg/Kg/d	After 1 year
Esomeprazole (Nexium)	0.7 – 3.3 mg/ kg/d	After 1 year
Rabeprazole (Pariet)	20 mg daily	12 – 17Y

Surgical treatment

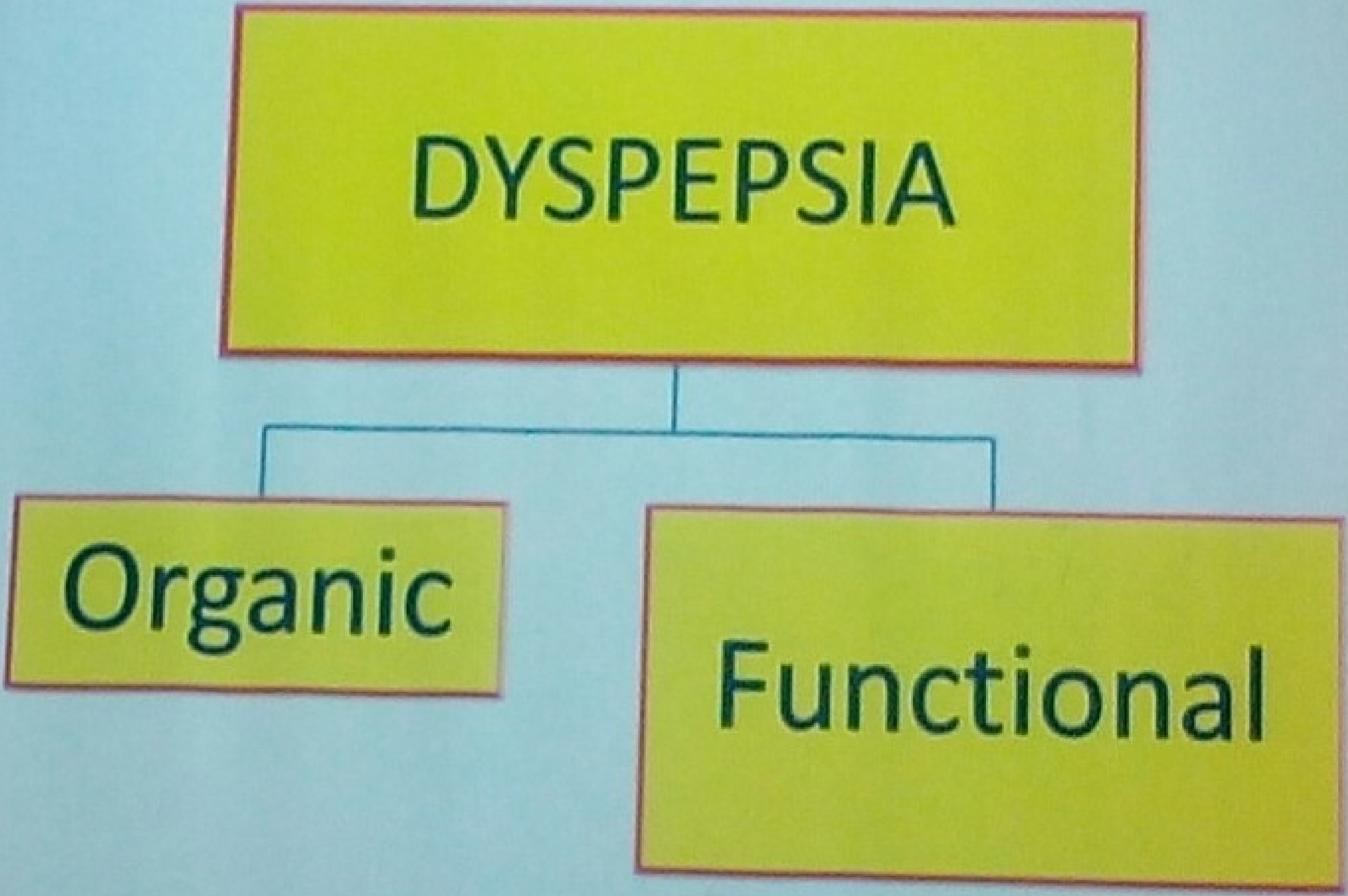
Niessen fundoplication in

1. Huge Hiatus hernia
2. Failure of medical ttt (at least 1-2 months).
3. Repeated aspiration and chest infection
4. Esophageal Stricture
5. Barrett change.
6. Patients with neurologic impairment.

A 6 years old girl presented with recurrent attacks of epigastric abdominal pain over the last 6 months.

Associated with vomiting; related to meals, no-bilious with no hematemesis. This was associated with, early satiety and postprandial fullness.

O/E: .... Mild epigastric tenderness, no organomegaly



DYSPEPSIA

- Organic
- Functional